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An Address.[1]

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President of the South Australian Branch of the British Medical Association.

IN accordance with the custom of this Branch, it is one of the privileges of the President on his retirement from office that he address members on some subject of his own choice. It is perhaps not unnatural that I should choose to speak on the control of tuberculosis, as this is a subject that has been of special interest to me for many years, and, despite recent activities, I am still far from satisfied with the progress made in building up an efficient organization to combat the disease.

[1] Read at the annual meeting of the South Australian Branch of the British Medical Association on June 24, 1936.

It is credited to the late King Edward VII that he said many years ago: "If this is a preventible disease, why is it not prevented?" The same remark would be equally applicable today. There are of course many difficulties, not the least of which are ignorance and apathy, and in these the patient is by no means always the only one at fault. Tuberculosis, and I refer in this address chiefly to the pulmonary form of the disease, is a condition concerning which we have much accurate information; but there are important gaps in our knowledge. We do not know for instance why it is that of those infected the majority throw off the infection and appear to suffer no ill effects, while others resist it badly, develop serious ill health and perhaps die as a result of it. It would be a great help in the campaign if we had some means of foretelling with reasonable accuracy which persons of those infected would overcome the infection unaided and which would not. The recognition of the fact of infection

is relatively easy, but the ultimate result of the infection is often impossible to forecast. It behoves us then to proceed with caution in the case of those people who are not quite well and are sensitized to the tubercle toxin, even though they present no definite clinical evidence of disease. The other great gap in our knowledge is our ignorance of some safe and easy method of eradicating infection even in its early stages, but more especially of overcoming well established disease. Undoubtedly cure, or if you prefer it permanent arrest, may take place; but only too often the tuberculous process resists all treatment, and persists for the remainder of the unfortunate victim's life.

On the other hand we know much concerning this disease. We know its cause, we know in a general way how it is transmitted and how to prevent that transmission, we know that it occurs in families, we are aware of the necessity of early diagnosis and treatment, and yet we do not make the progress in combating its ravages that we should. Figures show that the mortality rates are surely and steadily declining. This is a cause for satisfaction. But there is little real evidence that the amount of tuberculosis in the community has noticeably decreased. It would seem that the tuberculous patient is living longer, and so there are more alive in the community at any one time. And this is easily understood when we consider that most of our effort is directed toward the treatment of established cases rather than to prevention. In this State we have for advanced cases two sanatoria and a home, the sole function of which is treatment or amelioration. Moreover, the clinic established recently, after years of agitation, is at the present time almost wholly a treatment centre, few patients being referred for diagnosis. No great progress will be made while our vision is centred too narrowly on the established case: it should certainly include the home and any close contacts.

In this as in many other matters much depends on our philosophic outlook. If we believe that nothing really matters much, that it will be all the same in a hundred years' time, then we will not be unduly upset to know that each year some 250 of our young adults, many of them of the finest type, go to a premature death from pulmonary tuberculosis. If on the other hand we believe that we were brought into this world to pull our weight, to help ourselves and others to the utmost of our ability, then we will lend a helping hand and do something more than we are doing to put a stop to this tragic and preventible wastage of young lives.

For many years a good deal of thought has been given to this subject, and several reports have been written. But in this State at least the difficulty is to get thoughts and words transformed into acts and deeds. A very good report on the control of consumption was prepared for the Government so long ago as 1911 by the Chairman of the Central Board of Health at that time, Dr. Ramsay Smith. This followed upon a conference of delegates representing local boards of health held at Unley in 1909, and a departmental conference of the principal medical officers of health held in Melbourne in 1911. It is remarkable how clear and convincing that report is and how similar it is to our recommendations of today. But nothing was done. Tuberculosis, like the poor, is always with us: we accept it as a necessary evil and apathy descends upon us. Dr. Ramsay Smith concludes his report with the words:

> In setting forth these proposals, I have assumed that the necessary expense will present no difficulties. But if the matter of State economy, *i.e.* of wise management, should be raised, I would point out that, at the usually accepted and moderate estimate of the money value to the State of a human life, South Australia during the year 1909 was poorer by nearly £160,000 on account of deaths from consumption. If this had occurred once in a way from an epidemic of plague or smallpox, some attention would have been given to it. But it has been going on for years without much remark and with very little organized procedure for prevention. In thirty years the loss to this State totalled about £4,000,000.

Since that time the total has increased considerably and it is still mounting. The present Chairman of the Central Board in South Australia, in a report in 1932, said:

> As a community we are far too complacent. In our own small State, year by year, over three hundred people die from tuberculosis. They are mostly young adults, and this loss of valuable lives continues. It is a serious matter. If three hundred people in the prime of life were poisoned each year from drinking bad beer, would we remain so calm about it?

In 1929 Dr. M. J. Holmes prepared a splendid and most comprehensive report for the Commonwealth Department of Health. This investigation arose out of a recommendation of a Royal Commission on Health that the Commonwealth Department should formulate the principles of a comprehensive campaign against the spread of tuberculosis. To serve as a basis in formulating those proposals, a very thorough survey was made of the control of tuberculosis in each of the States. In March of last year, at a meeting in Canberra of the eighth session of the Federal Health Council, at which I was present by courtesy of the Council, Dr. Holmes's report was unanimously accepted as comprising a complete review of the subject. It is a report well worth the study of anyone interested. At the time the report was issued we fondly imagined that at last something was going to be done. But when the Commonwealth Government approached the State Government and was prepared to grant money for the purpose, it was politely told that control of health matters within the State was a matter for the State Government: in other words, that it should mind its own business. And so the matter lapsed again.

The first part of Dr. Holmes's report dealt with the position as it existed at that time. Concerning the position in South Australia, it stated, among other things:

> In South Australia the control of tuberculosis has not yet been placed on a definite systematic footing.
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> The Clinic does not as at present constituted undertake the ordinary systematic work usually associated with a tuberculosis dispensary.
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> There is no co-ordination in the existing organization for the control of tuberculosis. The tuberculosis problem is handled by two entirely separate and distinct departments between whom and other departments concerned no co-ordination seems to have been arranged.

Since that time improvements have been made in certain directions, notably in the provision of more and better hospital accommodation for the declared case and in the establishment of a tuberculosis clinic at the Adelaide Hospital. But it may still fairly be said that there is no coordination of existing facilities, and that much remains to be done in the matter of marshalling our forces and in their proper direction. Too much attention is still being focused on the declared case, and not enough on the detection and proper treatment of the incipient one.

In the second part of the report were discussed the most practicable measures to form the basis of a systematic campaign against the disease. Briefly the lines indicated were, on the one hand, the establishment of a system of fully developed dispensaries or chest clinics working in cooperation with institutions for the treatment of the disease; and, on the other hand, a well defined system for maintaining the nutrition of families of tuberculous patients, by improving home conditions, and by temporarily removing from the infected environment any one undernourished or showing presumptive evidence of having contracted infection which they were not resisting satisfactorily. The latter involved the establishment of "preventoria" especially for children.

Before discussing plans for the control of this disease, it is necessary to have a clear idea of the problem that confronts us, to know something of the nature of the disease and its distribution.

Nature of the Disease.

Although pulmonary tuberculosis has existed through the ages, it was not until a little over fifty years ago that Robert Koch demonstrated the causative relation to tuberculosis of a germ called the tubercle bacillus. And his demonstration was so complete that little of importance has since been added. With the discovery of the causative agent it was thought that the disease would soon be successfully controlled. But this has not proved to be the case, and although progress has been made, tuberculosis still transcends all other maladies in the total number of its victims and in the cost to civilized countries. The prevalence of tuberculosis is universal: no other disease is so widespread or produces so much poverty and long continued distress.

For tuberculosis to develop, there must be infection with the tubercle bacillus. This may gain an entrance to the body through its external covering, namely the skin, but much more frequently through a lining membrane as of the respiratory or alimentary tracts or of the tonsil. At the site of inoculation an inflammatory lesion occurs, and soon the tubercle bacillus passes to the lymphatic gland draining that area. At this stage there are few, if any, symptoms or signs of disease, and yet the tubercle bacillus has gained an entrance and very unpleasant results may follow. The frontier has been crossed and this may be the beginning of a lifelong struggle for that person.

The Distribution of the Disease.

Figures available in Australia tend to show that in every thousand persons about six hundred are infected with the tubercle bacillus. Of these about two hundred and fifty show lesions demonstrable after death, fifteen become clinically identifiable as suffering from the disease during life, and of every two thousand of the population one dies each year of pulmonary tuberculosis. It is quite clear that many people contract tuberculous infection which they overcome and which does them no apparent harm. On the other hand it is equally clear that many people develop serious disease which causes much invalidity and many deaths. For the last thirty years or more there have been in this State alone approximately from 250 to 300 deaths each year from pulmonary tuberculosis.

Prior to 1905 the death rate in South Australia was below that of Australia as a whole, but at about that time it arose above the rate for the whole Commonwealth, and it remained there until quite recently. When it is remembered that South Australia has an almost rural population as compared with some of the other States, that the climatic conditions are especially good, and that there is a striking absence of deleterious influences such as mining and large industrial concerns, the position in South Australia cannot be said to be satisfactory and it needs careful consideration. It is a striking fact that since notification was introduced there have been more than twice as many deaths from pulmonary tuberculosis as from all the other notifiable diseases combined. Here then is a very widespread infection, strong enough to make for itself a recognizable home in a quarter of the population, but faced with such a strong human resistance that less than one person in a thousand dies of it. As Dr. Cumpston, the Federal Director-General of Health, says: "On the one hand a ubiquitous enemy, striking here and there one in every four of us: on the other hand every individual a gallant and mostly a successful ally in the official fight against the disease."

Below fifteen years of age there is comparatively little mortality from pulmonary tuberculosis in Australia. But after fifteen the death rate rises steadily, reaching its maximum at from thirty-five to fifty-five years of age, after which it steadily declines until at or about the age of senescence cancer takes its place. In males tuberculosis is a mortal disease during early, middle and late adult life, but in females it is fatal mostly to young adults. One of the most serious aspects of tuberculosis is that it begins to make its presence felt in early adult life just when the individual has finished his education and when he promises to be of value to his family and to the State. With the supervention of tuberculous disease, instead of being an asset, only too often he becomes a burden to both.

The Type of Infection.

The incidence of bovine tuberculosis in the community is by no means inconsiderable, but the mortality among human beings from tuberculosis of bovine origin is low, having declined rather rapidly of recent years. This is particularly evident in the cities, and may reasonably be ascribed to improvement

in the milk supplies. Tuberculosis of bovine origin is seldom found except in children under the age of fifteen years and principally under the age of four. In most cases bovine tuberculosis appears to be limited to the lymph glands, bone and joint tuberculosis in Australia being usually associated with the human type of infection. Concerning the incidence of tuberculosis among dairy cattle in Australia we have little definite information; but, especially in the case of infants whose chief source of nutriment is milk, the possibility of their contracting bovine tuberculosis cannot be ignored. Part of the campaign against tuberculosis must be directed to a pure milk supply. The ideal would be milk from tuberculosis-free herds. But for economic reasons this is difficult of attainment, and a wise practical alternative would be to boil all milk used for infant feeding.

As Dr. Cumpston has pointed out:

> Tuberculosis of the lungs is human in origin—the decree may be made almost absolute. For all purposes of control it is the golden rule. In the industrial field one group of workers, namely miners, is exposed to a very special risk; but this risk is provided for as fully as possible. All else, the bovine and silicosis casualties apart, becomes a problem expressible in simple terms. There is constantly occurring the invasion along a known lymphatic route by a known bacillus of a fairly constant proportion of the people of Australia.

And although the majority of individuals successfully overcome this invasion, nevertheless there is a very considerable morbidity and mortality. Can this be lessened? A visitor from the Rockefeller Institute once said that we should have no tuberculosis in Australia. Granted the necessary powers and unlimited funds, perhaps this ideal could be more or less fully attained. But in this hard cruel practical world, neither the government nor the community generally is overburdened with money; so we must do the best we can with the resources at our disposal.

The Campaign.

The campaign against tuberculosis must be conducted on two main lines: (i) prevention of the spread of infection, (ii) early diagnosis and efficient treatment.

Prevention.

The prevention of tuberculosis from bovine sources has already been discussed. Prevention of tuberculosis of human origin is one of the particular functions of the central and local boards of health which are responsible for the administration of the *Health Act*. In the control of any infectious disease, notification must be the initial step. To be of value it must be followed by effective administrative measures. Within the limits prescribed by a comparative paucity of funds, very useful work is being done by the health authorities in educating sufferers and contacts in necessary preventive measures, and in the disinfection of premises after the removal or decease of the infective occupant. Regular visits are made by trained nurse inspectors, who supervise home conditions and disseminate useful knowledge. The value of the work would be increased if use could be made of these inspectors in persuading contacts to submit to critical overhaul. In the latter work the boards of health have been hampered in the past by the lack of arrangement for necessary diagnostic facilities. Now that the new clinic is completed and in proper working order, it is hoped this difficulty will be overcome. One of the most important functions of the clinic should be the examination of contacts and suspects referred to it for diagnosis.

In the work of prevention the private practitioner plays an important rôle, and he should receive every encouragement from the Health Department. He can help very materially in the education of the patient, who is likely to listen to no one so carefully as to his own doctor. He can also impress on contacts the necessity for thorough examination, and, in case of financial distress, he can direct them to the clinic for such examination. By close cooperation between the Health Department, the private practitioners, and the tuberculosis organization, valuable results are likely to accrue. In the public mind there is an undue fear of the infectivity of the tuberculous patient. Infection is conveyed almost solely by coughing and expectorating. Granted ordinary care and cleanliness, the consumptive carries no menace to other people. Risk of infection from table utensils is negligible if proper care is used in washing. If we were afraid of feeding and drinking utensils we should never again in our lives go into a restaurant or hotel. In very advanced stages of the disease, when the patient is too ill to worry any longer about preventive measures, as a rule it is best that he should be removed to an institution for cases such as his. The careless consumptive who persistently ignores the rules is a menace. Under an Act recently passed, power is given to deal with such a person. Wisely used, such an Act is a good one: the mere knowledge that it exists may be sufficient to call the erring one to a sense of his responsibilities.

But no matter how conscientiously instructions are given and obeyed, infection may pass, if indeed it has not already passed at the time of notification, to other members of the household or contacts. The prevention of the spread of infection is the ideal, but probably an ideal that will never be completely realized. After this the best we can do is to recognize which contacts have been infected and to help them to overcome that infection before dissemination and serious tissue damage have resulted. The systematic examination and supervision of contacts is an important aspect of any scheme for the control of this disease. Presuming that we shall never be completely successful in efforts to prevent the spread of infection, the detection and treatment of that infection in its earliest stages should be our aim. It is clear that many persons who become infected with the tubercle bacillus throw off the infection and never suffer from actual tuberculous disease in a clinical sense. Infection can occur without disease developing, but disease cannot occur without infection. The recognition of tuberculous infection is comparatively easy. But one of the great difficulties is to know which person will overcome the infection unaided or without special treatment, and which will develop disease and possibly succumb to it.

Early Diagnosis and Treatment.

There are many pitfalls in the diagnosis of early pulmonary tuberculosis. The difficulties arise from the facts that symptoms in many cases are quite lacking in distinction and there may be no abnormal physical signs. The old teaching of reliance on physical signs must be abandoned. An English physician, Hern, said recently:

> I believe that on balance more good than harm would result if it were possible to prohibit entirely physical examination of the chest for tuberculosis.

Symptoms may be so lacking in distinction that they fail to arouse suspicion in the mind of the observer: the absence of physical signs is still further misleading. To certain minds there appears to be something derogatory in being forced to admit dependence on special examinations. The plain fact is that special investigations are absolutely indispensable in the diagnosis of early tuberculosis; and the sooner this fact is realized the better. Such things as X ray examination, sputum examination and tuberculin tests must be carried out as a routine in addition to careful history taking and physical examination. Dr. Sinclair Gillies has cogently remarked:

> It is doubtful if the practitioner or the health authority fully grasps yet the paramount importance of early diagnosis and treatment.

Early recognition of the disease is of paramount importance, as treatment at this stage offers a good chance of cure. Once firmly established, the disease is very difficult to eradicate, and treatment only too often is unavailing.

Practical Measures.

In the campaign against tuberculosis the essentials are:

1. The appointment of a chief tuberculosis officer or director. He must be a specially trained whole-time medical officer paid by the State, and should be responsible for the organization of the whole scheme. The success or otherwise of the scheme will depend to a large extent on this individual who must have exceptional knowledge of the disease, unusual organizing capacity, and a wide outlook.

2. A tuberculosis dispensary or chest clinic. This will be the keystone of the organization and should be equipped to fulfil all the duties usually associated with such an establishment. It should have access to and be run in cooperation with the several special and general departments of a well equipped hospital. At its disposal there should be a sufficient number of hospital beds, in a special block set aside for the purpose.

3. Sufficient institutional accommodation for declared cases. (*a*) Sanatoria for patients with a reasonably good chance of recovery, and especially for those who cannot be effectively treated at home. (*b*) Hospital accommodation for intermediate cases to provide special treatment for those patients not well enough to be sent to a sanatorium, and not in so advanced a stage as to warrant their being sent to a consumptive home. (*c*) A consumptive home for advanced cases, where the patients can be made comfortable and given such treatment as befits their needs. The word incurable should not be used in referring to this home. It creates a bad psychological effect and increases the difficulty in persuading patients to go to it. There is no reason why these three, (*a*), (*b*) and (*c*), should not be run in conjunction. The hospital-sanatorium is the order of the day. (*d*) Suitable accommodation for patients with non-pulmonary tuberculosis, of bones, joints *et cetera* which often requires prolonged treatment.

4. Some practical scheme for the maintenance of the health and nutrition of the families of persons suffering from tuberculosis. In a long-drawn-out illness such as this frequently is, the economic factor is of vital importance to the affected individual. Some form of social service working in conjunction with the clinic is almost a necessity. We might devise some scheme of pensions or monetary help to assist the patient in the early stage when cure is practicable, rather than waiting as now until the disease is considered incurable before granting relief.

5. The establishment of homes or "preventoria" to which pre-tuberculous children or children showing any evidence of incipient tuberculosis could be sent for suitable periods for recuperation. Arrangements might also be made for the provision of camps at suitable times of the year to which children could be sent who, though poorly nourished or weedy, had not suffered in health to the same extent as those requiring more specialized care in "preventoria".

6. The provision of some scheme to utilize the work of the partially unfit, such as the establishment of industrial or farm colonies. The question of suitable employment for persons leaving sanatoria or after treatment is a pressing one. Only too often they are forced to return to unsuitable working conditions and promptly break down again. The consumptive, after arrest of the disease, needs to live a sheltered life for a year or two, or perhaps indefinitely.

7. More thorough education of students and if possible of practitioners in the subject of tuberculosis. In an already overcrowded university curriculum this presents some difficulties, which, however, should not be insuperable.

A scheme such as that outlined may seem a policy of perfection, and for economic reasons may not be possible of immediate attainment. But it is an ideal to strive for. The main parts of the scheme are already in existence; they need only proper organization.

The matters that I have spoken of are not new. They have been discussed over and over again and are now generally accepted as the basis of any scheme of control. Where we fail so sadly in this State is in having no controlling influence. Dr. Holmes in his report says:

> In order that the control of tuberculosis may be developed along efficient lines, it is considered essential that an officer should be appointed in whose hands would be placed the

co-ordination of all the activities associated with the control of the disease, and who would link up the administration of the legislation with the work of the treatment institutions and of the school medical service, and with the activities of a fully developed tuberculosis dispensary system extending out to the homes of all affected people.

This Association has approached the Government by report and deputation on the control of tuberculosis, and among other things has recommended the appointment of a whole-time chief tuberculosis officer. This recommendation was endorsed by the heads of both the Health and Hospitals Departments. But there has been no response. Later, when the matter of a reorganization of the Health Department was considered, among the recommendations made by this Association was one to the effect that a medical officer to control a tuberculosis division should be appointed. A carefully prepared report was forwarded to the Government, and this was followed by a deputation, but with the same result.

The inconsistency of our governments is amazing. They will cheerfully spend £100,000 on a home for patients with advanced tuberculosis, and then refuse to provide an adequately paid medical personnel. They expend huge sums each year on the care of patients with established disease, but cannot see that money spent on prevention is a better investment. It is strange that the average intelligent layman, when it is explained to him, can at once appreciate the fact that it is better to spend a relatively small amount of money on preventive measures than it is to continue to spend large amounts in a futile attempt to overcome established disease. But the politician seems totally unable to grasp this simple fact. When we approach him, he seems to think we are trying to put something over him. The Government raises an outcry at the idea of a cemetery being established at Northfield which it says would be "an unforgivable social crime" or "a proposal of the most callous nature". But a similar sentiment made no appeal to it whatever when it allowed a tuberculosis clinic to be established within a stone's throw of a hospital mortuary; and the fact that patients in a tuberculosis ward run sweeps on the number of corpses they see brought into the mortuary leaves it untouched. The Government will legislate for betting shops and appoint a commission to inquire into lotteries; but it turns the cold shoulder to a carefully considered recommendation, from an authoritative source, concerning the maintenance and improvement of the health of the people.

From the experience of the past few years it seems that deputations are politely received and reports promptly pigeon-holed. And that is all. It is not easy to see what can be done in these circumstances; but there is no need for despair. Time brings changes, even in five-year parliaments, and we may be permitted to hope that some day a government or a ministerial head will appear who will agree with us that, in dealing with a disease like tuberculosis, a little money spent in creating an efficient organization to prevent its ravages will be money well spent; and will do something about it.

NOTES ON SOME MODERN IDEAS IN HEART DISEASE.[1]

By R. WHISHAW, M.B., Ch.M., M.R.C.P.,
Hobart.

IT seems to be the usual custom to begin with an apology for the title of an address, but I do not think it is necessary here, for I hope that the following notes may be of interest to all of you. The notes have been collected from several hospitals and clinics, but I should like to pay tribute especially to the Heart Hospital, to the Cardiac Clinic of the London Hospital, to the inspiring teaching of Dr. John Parkinson and Dr. Evan Bedford, and to the fund of information to be found in the text-book of Dr. Paul White, of the United States of America.

FREQUENCY.

Heart disease has been estimated to occur in 2% of the population, and as a cause of death it heads the list, having outstripped tuberculosis, pneumonia and malignant disease. Here, then, is a good reason for discussing heart disease, for if these figures be applied to this town, there must be 1,200 sufferers from heart disease living among us.

ÆTIOLOGY.

The following English figures are indicative of the common types: rheumatism, 32% (45% to 50% in America); syphilis, 4%; hypertensive heart disease, 30%; arteriosclerosis, 25%; thyreotoxicosis, 3%; congenital heart disease, 1% to 2%; *cor pulmonale* (chiefly due to emphysema), 0·5%; and bacterial endocarditis, acute and subacute, 5%. I cannot find any Australian figures, but I imagine that the figures dealing with rheumatism would be considerably lower and those of thyreotoxicosis perhaps higher.

Rheumatic Fever.—Since the recent work at Great Ormond Street there is a more general feeling that rheumatic fever may be caused by a virus and that the virus lowers the victim's resistance to the secondary invader, which is the streptococcus. With very few exceptions, such as acute bacterial endocarditis, acute rheumatism is now regarded as the sole cause of all mitral lesions, including those which occasionally follow scarlet fever, the joint pains in the latter disease being thought to be due to coincident rheumatic infection. Mitral stenosis with no rheumatic history is nevertheless due to this infection, without joint involvement, a not uncommon occurrence in children.

Diphtheria.—Diphtheria causes no chronic heart disease. The exotoxin or toxoid, of course, is responsible for acute symptoms and death, which is due to various types of heart block or to vagal or splanchnic paralysis—peripheral vasomotor failure.

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on April 21, 1936.

Pneumonia.—In pneumonia death may occasionally be caused by right heart failure, but it is much more frequently due to general peripheral vasodilatation and failure, so that the heart receives no blood to pump.

Examination of a Patient with Heart Disease.

The routine examination of a patient with heart disease is accomplished in four stages in every case. These stages are: (i) history-taking, (ii) physical examination, including estimation of the blood pressure, (iii) electrocardiographic study, (iv) examination under the fluorescent screen.

History-Taking.

Taking these in order, briefly, we can dismiss the history-taking with a few words, important though it is. Inquiry should always be made as to when the heart was first known to be affected, how the present condition compares with the past, and what treatment has been given. Neglect of these obvious questions may lead, for example, to a mistaken diagnosis of rheumatic endocarditis, when the lesion is congenital. Inquiry should also be made with regard to past illnesses known to be connected ætiologically with heart disease, the important ones being rheumatic fever and chorea, recurrent tonsillitis, scarlet fever, syphilis, hypertension, thyreotoxicosis, nephritis and chronic lung diseases, especially emphysema.

Physical Examination.

Nothing has superseded the time-honoured quartet of inspection, palpation, percussion and auscultation.

Inspection.

With regard to the first, I would merely remind you to examine the neck for four signs: (i) Distension and abnormal pulsation of jugular veins, especially the right, when the patient is sitting up. This is important in congestive heart failure and in obstruction to the superior *vena cava*. (ii) Vigorous arterial pulsation. This is distinguished from venous pulsation by palpation—venous pulsation is not palpable. The former occurs in aortic regurgitation, aneurysm, thyreotoxicosis, and chronic hypertension. In hypertension you must often have noticed on the right side such a vigorous pulsation or expansile swelling as to suggest aneurysm. This is due to the "unwinding" of the aorta and the raising of the arch, thus kinking the carotid artery, which is fixed at each end. (iii) An enlarged thyreoid gland. (iv) Tracheal tug.

Palpation.

Palpation is useful in finding the maximal apex impulse, and occasionally in finding out the extent of its shift with change from the left to the right lateral position, which is normally five centimetres. Absence of shift suggests pericardial adhesions. Thrills are not uncommon when felt for as a routine. Systolic thrills are commonest in the second interspace, either to the right or the left of the sternum, being due to aortic stenosis or aneurysm. On the left, a little lower down, is the site for pulmonary stenosis and patency of the *ductus arteriosus*. Lower still, interventricular septal defects are the most likely cause. Diastolic thrills are usually due to mitral stenosis. If there is doubt about the presence of a thrill, it can often be made obvious by gently exercising the patient and then examining him at he leans forward and holds his breath after a deep expiration.

Percussion.

Percussion to determine the size of the heart has now been abandoned by many leading cardiologists. It is useful in the diagnosis of pericarditis with effusion, especially percussion in the upper two interspaces and sternum, where also aortic disease may give relative dulness, and in other mediastinal diseases. Percussion is most important, of course, in examination of the lungs, which should be part of the routine in the examination of every cardiac patient, in order to determine the presence of hydrothorax (commonly right base) and chronic pulmonary disease, which may cause heart displacement and which may give a false impression of enlargement. This occurs in such conditions as lung collapse, chronic fibrosis and pneumothorax, as well as fluid. I need not remind you that the feeling of resistance is often quite as important as the relative pitch of the percussion note.

Auscultation.

Before discussing heart murmurs I should like to draw your attention to the importance of detecting gallop rhythm. This consists of the occurrence of three distinct heart sounds, and must be distinguished from reduplication or splitting of the heart sounds. The commonest type of gallop rhythm is when the third heart sound follows the normal second, which is known as protodiastolic gallop rhythm. It is rarely found in mitral stenosis, in heart block and in neuro-circulatory asthenia; but commonly, and it is here where its importance lies, it occurs in early left ventricular failure in such conditions as chronic hypertension, coronary and aortic disease. Its presence in such cases carries a bad prognosis, for death may occur within a few weeks or months of its appearance, but with careful treatment may be postponed for a year or two.

Murmurs.—During the last century every murmur heard was thought to indicate heart disease. When most of us were students the pendulum of opinion swung rather too much the other way, and at the present time an attempt is made to interpret them correctly. We can only touch on the question of murmurs tonight and point out that diastolic are much more important than systolic murmurs, the latter so often being functional and not due to organic heart disease. Nevertheless they must always be carefully investigated; and it is important to remember that small valvular defects with rapid blood flow cause loud murmurs, while large defects and slow blood flow may cause no murmur or only

a faint, soft murmur, not easily detected. This is likely to be so when the heart fails. Also, as you know, such serious diseases as hypertensive heart disease, coronary disease, aortitis and serious congenital defects may be present without murmurs, and even the heart sounds may be normal. A loud murmur, then, does not necessarily indicate serious valve damage and in itself not even organic disease.

A systolic murmur at the apex is not now regarded as evidence of mitral regurgitation; but when it is present with a history of acute rheumatism and other signs of endocarditis, it is labelled as indicating mitral valve disease or stenosis. *Post mortem* evidence is responsible for this view, regurgitation in the absence of left ventricular hypertrophy never being present without stenosis.

Functional systolic murmurs are very much more common and may be due to a variety of factors, such as relative mitral incompetence resulting from left ventricular enlargement, cardio-respiratory effects, and conducted murmurs from elsewhere. In old people an apical systolic murmur is often due to aortic disease. Congenital septal defects also have to be considered.

Diastolic murmurs at the apex are most often due to mitral stenosis and occur any time during diastole; they do not necessarily occur only in late diastole. Previously late diastolic murmurs were known as presystolic murmurs. You all know the low-pitched rumble with presystolic accentuation of mitral stenosis, with an accentuated first heart sound, and its localization to a small area. When this murmur is faint or absent it can often be brought out by gentle exercise or by administration of a nitrite to increase the speed of blood flow. An error is sometimes made in mistaking for mitral stenosis the roughening or accentuation of the normal first sound which occurs in tachycardia from any cause. Other methods of confirmation or otherwise, to be discussed shortly, are available in doubtful cases. Another pitfall is the conducted diastolic murmur of aortic incompetence. If this murmur is due to syphilitic aortitis or atheroma, it is known as the Austin-Flint murmur and mitral stenosis is not present, as neither of these diseases affects the mitral valve.

Systolic murmurs at the base are commonly due to one of the following four conditions: (i) aortic stenosis, (ii) dilatation of the aorta without aneurysm, (iii) aortic aneurysm, (iv) transmission from the pulmonary area. Dilatation of the aorta without aneurysm is the commonest and may be due to luetic aortitis, chronic hypertension or atheroma. A faint systolic murmur over the pulmonary area is often normal and is due to physiological dilatation of the pulmonary artery. A harsher murmur is less common and may be due to congenital pulmonary stenosis, patent *ductus arteriosus* (when it may assume a continuous character, with systolic accentuation, the "humming top" murmur), or coarctation of the aorta. Congenital interventricular septal defects (Roger's disease) also may produce harsh murmurs to the left of the sternum, but are usually most pronounced at a lower level—in the third or fourth space. Aortic stenosis is diagnosed on the triad: murmur, thrill and small plateau type of pulse, with low pulse pressure. The second sound is often absent.

Diastolic murmurs at the base are most important and sometimes difficult to detect. The murmur of aortic regurgitation is often low and very soft, and is quite as often found to the left as to the right of the sternum. This soft blowing diastolic murmur is best picked up with the bell type of stethoscope, and the patient should be leaning forward and be asked to stop breathing after an expiration. If the murmur is still inaudible, the naked ear applied to the chest sometimes succeeds when the stethoscope fails. When the murmur is loud, it may be widely transmitted and heard at the apex, mid-sternum and over the vessels of the neck. The causes of aortic regurgitant murmurs are: (i) organic aortic valvular disease due to rheumatic infections, luetic aortitis and atheroma; (ii) rarely, dilatation of aortic valve ostium as the result of aortitis, chronic hypertension and severe anæmia.

The other signs of aortic regurgitation, such as collapsing pulse with full pulse pressure (the diastolic pressure often being down as low as 30 to 40 millimetres of mercury), the capillary pulse (best seen with a microscope slide pressed against the lower lip), are all signs of severe disease, and about 50% of early cases will be missed unless the soft blowing murmur is most carefully looked for. When the full clinical picture is developed, the water-hammer pulse is obvious in all superficial arteries, as well as being palpable at the wrist. The best way to detect this clinically is to hold the arm well up, with your left hand lightly round the arm, palpating the brachial artery, while the right fingers are across the radial.

The heart bears the strain of aortic stenosis much better than it does that of regurgitation. In both the left ventricle will be hypertrophied. It is a good plan to get into the habit of palpating both pulses. I have seen several cases in which absence of or feeble pulse in one wrist gave the clue to the diagnosis of aortic aneurysm, the latter partially occluding the mouth of the subclavian or innominate artery. An aberrant radial artery must be excluded by observing whether pulsation is also absent from the brachial artery. Before leaving this subject I should like to remind you of the importance, first, of detecting early aortic incompetence, and, secondly, of finding its causation and, if in doubt, of having a Wassermann test done. Few patients with syphilitic aortitis live longer than five years, but often, especially if the condition is detected early, much can be done with specific remedies.

Radiology of the Heart.

The development of the radiological part of the physical examination has now reached the point at which it is used as part of the routine. Every new patient is examined under the screen, and occasionally an orthodiagram, that is, a tracing from the

screen, is made and filed for future reference. Only by radiology may the size and shape of the heart be ascertained with certainty; hence the remarks on percussion made previously. Owing to the great variation of normals, actual measurements are not much used. The cardio-thoracic ratio is, however, sometimes useful to determine whether slight degrees of enlargement are present. This is the measurement on the screen of the transverse diameter of the heart divided by the internal diameter of the thorax, the average, in round figures, being 50%.

Sometimes surprising and often valuable information is obtained by routine fluoroscopy. It is unfortunately of no value in early cases, and serious disease may be present without radiological evidence. The patient is examined in three positions: first, in the antero-posterior, with his chest against and exactly parallel with the screen and with the tube behind; secondly, in the first oblique position, with the patient turned half left; and, thirdly, in the second oblique position, with the patient turned half right.

When a normal heart is examined, the inferior border is seen to be obscured. It shows up in only two conditions: (i) In pneumo-peritoneum, such as that which occurs in ruptured gastric and duodenal ulcers. This is a useful method of confirmation or otherwise, when used as a routine in suspected cases. (ii) In that very rare condition in which the colon is interpolated between the heart and the liver.

On the right border the following structures, from above downwards, may be seen: superior *vena cava*, convex edge of the ascending aorta, the right auricle, the inferior *vena cava* (sometimes).

On the left border the following are seen: aortic arch, pulmonary artery and infundibulum of the right ventricle, and the left ventricle, which occupies a narrow strip along the lower two-thirds. The right ventricle occupies the greater part of the anterior surface.

It will be noted that the left auricle does not normally appear on the surface; it is completely behind the other chambers and should have been called the posterior auricle (Parkinson).

Three normal heart shadows must first be recognized: (i) the average; (ii) the horizontal heart, which is shorter and wider, and occurs in stout people with a high diaphragm; and (iii) the vertical heart, in people with long, thin chests and low diaphragms; they also have long, thin hearts.

In the first oblique view the right side of the picture is composed, from above downwards, of the ascending aorta, the bifurcation of the pulmonary artery, pulmonary artery and right ventricle. The left side, from above downwards, consists of the descending aorta, left auricle, right auricle and inferior *vena cava*. This view is especially useful in estimating the size of the left auricle, also the width of the aorta. When the œsophagus is filled with a thick emulsion of barium it can clearly be seen descending between the left auricle and aorta. The indentations observed above are caused by the crossing of the aorta and bifurcation of the trachea (or left bronchus). Below this is a gentle curve following the outline of the left auricle. In mitral stenosis this curve is much more pronounced and in some cases becomes extreme, so that the shadow passes over that of the spine or even beyond, and is caused by the left auricular enlargement. The antero-posterior view of mitral stenosis is also characteristic, showing the enlargement of the right side of the heart, the prominence of the conus of the right ventricle (on the left border) and the pulmonary artery, and the congestion of the lung roots ("cherub's wings").

The second oblique view is useful in studying the aortic shadow, as the vessel can be seen almost in its entire intrathoracic course. Normally the ascending aorta, above the heart shadow, should not project beyond it, nor beyond the spine behind. In aortitis, in aneurysm, and sometimes in hypertensive heart disease and atheroma the aortic shadow bulges beyond these limits. In this view the left auricle and left ventricle lie on the side towards the spine, while the right auricle and ventricle make up the opposite border. An aneurysm causes damage to surrounding structures according to its situation, of course. A very large aneurysm may cause a little damage, while a small one may occlude a bronchus, with resulting atelectasis of the corresponding lobe. If the atelectasis is unrelieved the whole lung may collapse, and collapse may be followed by unilateral fibrosis of the affected lung. The clinical picture may then be one of unilateral fibrosis, with displacement of the heart to the affected side. Clinically this may be difficult to distinguish from the much more common causes of the condition—bronchial carcinoma and fibroid phthisis. In such cases, therefore, it is wise to examine the heart carefully for signs of aneurysm and to have a Wassermann test carried out before a bronchoscopic examination is performed, bronchoscopic examination being a dangerous procedure in the presence of intrathoracic aneurysm. Speaking of displacements of the heart, it is important to remember that scoliosis has this effect on the heart, the heart fitting into the concavity of the curve and giving a false impression of enlargement. Severe scoliosis with considerable displacement may eventually lead to cardiac failure on account of the long-continued strain.

In aortitis the antero-posterior view shows widening of the base shadow, and left ventricular enlargement if aortic regurgitation is present. In the first oblique view, the first indentation of the œsophagus will be marked and the widening of the aorta again observed. If the heart is much enlarged, the auricular curves may be deepened. In chronic hypertension *plus* atheroma, as well as in luetic aortitis, the aorta "uncoils", and on the screen may be seen to take a wider curve than normal, both to the right (ascending) and left (descending). This so-called uncoiling is usually more even in

atheroma than in aortitis. Chronic hypertension is thought to be due to peripheral arteriolar spasm of unknown origin. This spasm leads eventually to diffuse sclerosis of the arterioles in various organs. The sclerosis is the result of the spasm, and not its cause, as the high pressure is present long before the sclerosis appears. Atheroma, which affects the larger vessels, and especially the aorta, is a disease quite apart and does not depend on spasm and high pressure. Nevertheless, long-continued hypertension predisposes to early atheroma, so that the latter is apt to appear in younger people. This can sometimes be seen in skiagrams, the calcified plaques appearing in the aortic wall.

Some congenital abnormalities throw characteristic shadows. The most striking perhaps is coarctation of the aorta. Here the heart is usually enlarged (left ventricle), with a prominent pulmonary artery and disappearance of the aortic knuckle. The most interesting observation, however, is erosion of the ribs (Rŏstler's sign), due to the enlarged and tortuous intercostal arteries, which help to support the collateral circulation. The site of the stenosis of the aorta is in the vicinity of the insertion of the *ductus arteriosus*; and when the stenosis is well marked, it gives rise to increased blood pressure above the stenosis. For example, in the brachial arteries the systolic pressure may be 200 millimetres of mercury or more, while in the femorals it may be 100 millimetres or less. The collateral circulation goes by way of the internal mammary, scapular and intercostal arteries. These enlarged vessels may often be observed in physical examination of the chest. Long systolic murmurs are sometimes heard over the chest and along the course of the dilated anastomotic vessels.

In congenital patency of the *ductus arteriosus* the pulmonary artery shadow is often enlarged to a considerable degree and pulsates vigorously on the screen. Dextrocardia is often a complication. Two types are recognized. The first consists merely of rotation of the heart to the right, in itself causing no symptoms, but nearly always associated with some other cardiac abnormality, such as patency of the *ductus arteriosus*. The second is the so-called "mirror" type, with complete transposition of all chambers and usually the abdominal viscera as well. This can be seen on the screen, and the gas bubble in the stomach will be on the right instead of the left side. In patency of the *ductus arteriosus* there may or may not be present the characteristic "humming top" type of continuous murmur, with systolic accentuation, best heard in the second left interspace. Apart from this, it may be distinguished from mitral stenosis by the absence of the increase in the left auricular shadow in the first oblique position (after the swallowing of barium).

Congenital dextro-position of the aorta may sometimes be seen on the screen. After the taking of barium the shadow in antero-posterior views shows the œsophagus passing to the left, instead of the right, as it is crossed by the aorta. This observation, of course, is not proof of the existence of the condition. The condition is incompatible with life unless there is a coexisting septal defect, such as a patent *foramen ovale*, or interventricular septal opening to allow some of the venous blood to reach the lungs. The common combination, and one that occurs in 75% of adults with cyanosis due to congenital *morbus cordis*, is that known as Fallot's tetralogy. The four components of the tetralogy are: dextro-position of aorta, pulmonary stenosis, interventricular septal defect, and hypertrophy of the right ventricle. This gives the sabot-shaped or clog-shaped heart, the "instep" being due to absence of the pulmonary prominence, and the "toe" to right ventricular enlargement. Occasionally the pulmonary stenosis is replaced by dilatation, which can be seen well on the screen, while the other components are also present (Eisenmenger's tetralogy). More rarely still, the dilatation of the pulmonary artery may be the sole defect. More commonly it is associated with patency of the *ductus arteriosus*. Lesser degrees of dilatation of the pulmonary artery are not uncommon in (non-congenital) chronic lung diseases, especially emphysema (*cor pulmonale*), and in the increased pulmonary tension that occurs in mitral stenosis. The congenital cases are often so pronounced as to give some relative dulness on percussion over the pulmonary artery, with pulsations in the second and third interspaces and harsh systolic murmurs.

Before leaving the question of congenital heart disease, which we have touched on, I want to mention two points of practical interest. The first is that in septal defects of smallish size there may be no symptoms until, perhaps after adult life has been reached, for some reason, such as an infection, or for no obvious reason, the direction of the circulation is reversed. There is a venous shunt and insufficient blood reaches the lungs, so that cyanosis and dyspnœa are observed.

The second point is merely to remind you of the frequency of lung infections in congenital heart disease, especially tuberculosis. Tuberculosis is often the cause of death, the other common causes being congestive failure and bacterial endocarditis. The latter is especially liable to occur in valve cusp defects, such as bicuspid valves.

Other conditions in which radiology sometimes gives considerable help are: calcification of the pericardium, which occasionally follows a pericarditis from any cause and which may be relieved surgically; and aneurysmal dilatation of an auricle as a complication of mitral stenosis. This finding may solve the problem of an obscure laryngeal paresis due to the elevation of the left bronchus with pressure on the recurrent laryngeal nerve. Lastly, an aneurysm of the left ventricle may prove the presence of a coronary thrombosis. Speaking of coronary occlusion, I would remind you that, although the classical signs have been so much stressed recently that all are on the alert, quite a high percentage occur with little or no pain, and the signs are only those of congestive failure, cardiac

asthma, or acute pulmonary œdema. A small vessel becoming occluded may give rise only to attacks of faintness with no pain, and sometimes apparently without clinical symptoms.

The electrocardiograph shows some changes in most cases, though sometimes not for some hours, and occasionally not for a day or two.

I have merely indicated certain conditions in which radiology is helpful in the diagnosis of heart disease, and I am conscious of equally important studies unmentioned, but hope that sufficient has been said to prove the value of this form of examination in cardiology.

Electrocardiography.

I shall not spend any time on the important subject of electrocardiography, but its importance must be appreciated, as an electrocardiogram is now an essential of every cardio-vascular examination. The correct interpretation of a tracing requires considerable experience and study. It must be clearly realized that the tracing may be normal in the presence of severe heart disease; but more often it gives valuable help when read in conjunction with the clinical findings, and in some cases may be the only method of arriving at a correct diagnosis. Examples of the last mentioned are certain cases of coronary thrombosis. Most types of arrhythmia are difficult or impossible to diagnose clinically, for example, auricular flutter, frequent premature beats and interpolated premature beats simulating auricular fibrillation. Also there are the various types of tachycardia, of heart block, including bundle branch block, the bigeminal pulse or coupling, all of which require a tracing for their solution. Electrocardiography is also useful in watching the effect of digitalis and of thyreoid therapy in myxœdema, and in estimating the preponderance of either ventricle and the state of the myocardium. The electrocardiograph is therefore not merely of scientific interest, but frequently affords the only clue to correct diagnosis and treatment.

Diagnosis of Heart Disease.

A diagnosis of "heart failure" or "aortic regurgitation", and so on, is now regarded as quite inadequate. The diagnosis must include the ætiological, the structural and the functional factors. In heart failure, for example, it must be stated whether the failure is of congestive or of peripheral type; and an attempt is even made to decide whether the failure is due primarily to the right or left ventricle, although this may be possible only early in the case. Common causes of right ventricular failure are mitral stenosis and pulmonary emphysema; and of left ventricular failure, chronic hypertension and aortic insufficiency. Circulatory stasis may primarily be in the lungs owing to failure of the left ventricle, and in the liver and dependent parts in right ventricular failure. Vague terms, such as "dilatation of the heart", "myocarditis", "heart strain", have no place in cardiology.

I have already discussed the ætiological and functional factors, and the structural factor will be revealed on physical examination, so that a full diagnosis can be made. An example is rheumatic heart disease (ætiological) with mitral stenosis (structural), auricular fibrillation and congestive failure (functional). Complications, if present, are added, and it is often useful to mention actual physical capacity. A simple classification (White) is:

1. Full normal activity without cardiac symptoms.
2. Activity, slightly, moderately or greatly restricted by symptoms.
3. No activity possible without symptoms.
4. Symptoms even at rest.

As opposed to congestive failure is the type known as peripheral or general vascular failure. General vaso-dilatation is responsible, and is sometimes due to liberation of a histamine-like substance by the damaged tissues as in trauma, or to toxins as in the peripheral failure of pneumonia.

Before leaving the question of congestive failure, three other important clinical conditions must not be forgotten. These are gallop rhythm (which we have already noted as an ominous sign), *pulsus alternans* and cardiac asthma.

Pulsus Alternans.—Except when the pulse is very rapid, when it may be ignored, *pulsus alternans* indicates a failing heart, and if well marked spells death in a few months at most. It consists of alternating large and small pulse waves with normal rhythm, and often appears before the signs of congestive failure. In early cases it frequently follows a premature beat for several cycles, but later may be constant. It is most easily discovered during blood pressure reading. At the moment of registering the systolic pressure it is noticed that the pulse rate is halved, without arrhythmia to account for it. On lowering the pressure a few points, the normal rate returns. When very well marked, *pulsus alternans* may be detected by the finger on the pulse.

Cardiac Asthma.—Cardiac asthma is a distressing and important condition associated with left ventricular failure. It occurs most often at night; and the patient is wakened by severe dyspnœa of definite asthmatic type, which may end in pulmonary œdema and death. Usually, however, the attack is less severe and passes off after an hour or so. Mild attacks occur at any time and may go unheeded and undiagnosed. Cardiac asthma is found in those diseases causing left ventricular hypertrophy, such as chronic hypertensive heart disease and aortic disease. The same may also be said of *pulsus alternans*. The duration of life after the first attack of cardiac asthma averages sixteen months.

Functional Diseases of the Heart.

The most important functional disease of the heart is neuro-circulatory asthenia, the effort syndrome or autonomic imbalance. It is an important and often neglected condition which I should like

to mention before going on to therapeutics. It occurs in 10% of all heart diseases and is a complication of about 3% of organic heart disease; it was well known to you all as "D.A.H." during the War. It is part of a neurasthenic state and is often labelled cardiac neurosis *et cetera*. It is important to recognize it, not only because it complicates other diseases, but because it is a disabling condition, if neglected, and with early treatment may be cured. It is a functional condition, with no pathological lesion. In sensitive people it may follow nervous shock, anxiety, emotional conflicts, worry and so on; and it is not uncommon in normal individuals after an illness such as influenza or after severe fatigue. It is commonest in young adults, and there is often a family history of nervous instability.

The symptoms are submammary pain and palpitation and dyspnœa, and in severe cases, faintness, dizziness, tremors, sweating and nervousness. The pain is non-anginal and often comes on at night or some hours after exertion, indicating that it is not due to coronary ischæmia, for, as Parkinson says: "The heart does not remember." The dyspnœa is mostly subjective and frequently sighing, the latter being almost pathognomonic of this disease. The symptoms are aggravated by excitement, exertion and fatigue, but gentle exercise often relieves them.

On examination the heart is rapid and rather forceful, but otherwise normal (unless the condition is a complication of organic heart disease, which is not uncommon). There is often tremor, worried expression, flushing and sweating, and often slightly raised blood pressure—in other words, a syndrome closely resembling thyreotoxicosis. The absence of a palpable thyreoid and of eye signs by no means clinches the diagnosis, as so-called "masked" hyperthyreoidism is not rare. A careful estimation of the basal metabolic rate is necessary in all doubtful cases, for incipient tuberculosis may give a similar picture.

Speaking of thyreotoxicosis, I was most interested to compare the results of thyreoidectomy in two separate clinics. In one the results were uniformly good and in the other a percentage of the patients failed to recover after operation. In the former clinic the basal metabolic rate was estimated as a routine, and in the latter not at all. The failures, or at any rate most of them, occurred in cases of neuro-circulatory asthenia. Admittedly there are borderline cases in which the estimation does not solve the problem, but in such cases the administration of iodine for a week or ten days followed by another estimation is quite conclusive. The clinical iodine test, without control, is unsatisfactory. The basal metabolic rate is also useful in deciding the best time for operation in thyreotoxicosis and in checking the post-operative course. It is also very helpful in cases of subthyreoidism, myxœdema and cretinism, both in diagnosis and treatment.

To return to neuro-circulatory asthenia, we have still to discuss the treatment. The result of this depends a great deal on the care with which the physician explains the cause of the trouble and the reassurance and suggestive help that he gives. To tell the patient that there is nothing wrong is just as bad as making a diagnosis of heart disease, and the result may be, and often is, permanent incapacity. This preliminary is the most important part of the treatment. The patient's mode of life must then be discussed and regulated as far as possible; he must avoid late hours, excitement and strong tea; tobacco and alcohol must be taken only in moderation, and so on. Mild sedatives at first are a help, but should soon be given up; and digitalis should never be given. To clear up a septic focus often helps, but an infection nearly always causes relapse. Reassurance and reeducation are the keynotes to success.

THERAPEUTICS.

Firstly we shall discuss "heart attacks", so-called, and their treatment in the relative order of their frequency.

Angina Pectoris.

The treatment of an attack of *angina pectoris* may be summed up in one word: trinitrin. This is by far the best, and should be given in tablet form and chewed. The action is then many times more rapid than when the drug is swallowed. This was proved by a person suffering from angina, who had a gastrotomy tube. His attack quickly subsided when he chewed a tablet, but when one was placed in his stomach direct it had a delayed and unsatisfactory effect. In preventive treatment, besides the usual measures to secure rest *et cetera*, trinitrin is also very useful. A tablet is taken before any slight necessary exertion, such as taking a bath, or before any effort that usually causes an attack. Within reason, the number of tablets taken daily is unrestricted; up to thirty or more cause no unpleasant after-effects. Intravenous injections of glucose in a 1% to 20% solution (gradually increased), with or without five units of insulin, is often of value. It may be repeated three times a week for several weeks. In desperate cases and in those in which severe pain comes even when the patient is at rest, the paravertebral injection of three to five cubic centimetres of 85% alcohol into the upper five dorsal *rami communicantes* must be considered. The injection is made four centimetres from the mid-line of the back; and 50% to 75% of patients are reported to be completely relieved by this treatment. By preventing the warning symptoms of ischæmia, however, more exertion is likely to be taken, often with fatal results; moreover, the after-effects are sometimes painful. Deep X ray therapy is also reported to be successful in some cases, as is thyreoidectomy.

Paroxysmal Tachycardia.

Paroxysmal tachycardia may be regarded as a series of premature beats, and of the three types auricular tachycardia is by far the most common and is usually a functional condition. The rhythm

is regular and the beats average 170 per minute; the onset and offset are sudden, and the condition lasts a few minutes to a few hours. The nodal type is rare and the ventricular type is always serious, as severe myocardial damage is its cause; it often results from coronary occlusion and is frequently a terminal event. It is extremely seldom that congestive failure occurs purely as a result of tachycardia, but it does occur when some underlying organic disease is present. The treatment of the common (or auricular) type is, first, reassurance. An attack can sometimes be stopped by carotid pressure on the right side or by pressure on the eyeballs or by certain postures. Bromides are useful in prevention. In a long-continued attack quinidine may be successful; it may be tried in preventive treatment; digitalization may also be tried. Certain factors which excite attacks should be sought for and eliminated, such as constipation, over-eating, too much tea and tobacco. The condition has to be distinguished from normal sino-auricular tachycardia, auricular flutter and fibrillation, by its sudden onset, regularity and short duration. An electrocardiogram is necessary if one wishes to be certain (also for diagnosis of the type).

Cardiac Asthma.

I have already discussed cardiac asthma. The treatment consists in absolute rest and morphine, which may be repeated, and if necessary later, venesection. Intravenous administration of dextrose is useful in prevention, and the patient must be digitalized and kept so.

Coronary Thrombosis.

As death in coronary thrombosis is frequently due to ventricular tachycardia, all patients are given quinidine as a routine in Germany, but not in England.

Embolism may follow coronary occlusion, and the liability may be increased by quinidine, quite apart from its toxic effects. Morphine, 0·06 gramme (one grain) or more, is given at the onset in divided doses, and it is necessary to give enough to afford relief. For failure and auricular fibrillation, which often follow, digitalis is given, but with caution; for collapse, caffeine and ephedrine. Remember that a large heart with a low systolic and high diastolic blood pressure is suggestive of infarction, and that the condition may be obscured by acute pulmonary œdema, cardiac asthma or fibrillation and failure.

Acute Pulmonary Œdema.

Acute pulmonary œdema occasionally complicates chronic hyperpiesia, aortic and mitral stenosis. Sputum is salmon-coloured, but the colour may be absent, and the characteristic sign is the profuseness of the expectoration. The onset is very sudden, with pallor, then cyanosis and suffocation. The treatment is by venesection, followed by morphine with atropine, and perhaps intravenous administration of digoxin or strophanthin, 0·26 milligramme (one two-hundred-and-fiftieth of a grain), and oxygen inhalation.

Pulmonary Infarction.

Pulmonary infarction may be due to thrombosis or embolism, usually the latter, the embolus coming from the right auricular appendix in auricular fibrillation or from peripheral thrombo-phlebitis due to various causes, including operation. If the occlusion occurs in a large vessel, the onset is very sudden, with pain in the chest, extreme dyspnœa, rapid distress and cyanosis and death in a few minutes. When smaller vessels are involved there is sudden pain and dyspnœa, and later cough develops, with in some cases blood-stained sputum and pyrexia. The signs and symptoms strongly suggest pneumonia. The X ray shadow is ovoid, not sharply defined, and not typically wedge-shaped, as one would expect, and it usually disappears fairly soon, unless fibrosis occurs. It is commonest at the right base. Treatment is by oxygen administration, morphine and venesection, and stimulants for collapse.

Hypertensive Encephalopathy.

Hypertensive encephalopathy is not a heart attack, but is conveniently considered here. This clumsy name refers to attacks of cerebral arterial spasm, followed by dilatation and œdema, which occasionally occur in hyperpietics and nephritics. The blood pressure is always raised. The same syndrome occurs in acute lead poisoning (now hardly ever seen) and in eclampsia. There is no nitrogen retention, and the condition is distinct from uræmia. The attacks come with severe headache, perhaps convulsions and coma or mental changes, or with sudden loss of vision. The veins are prominent in head and neck, and there may be neck rigidity, tachycardia and slow respiration. Sometimes there is hemiplegia or monoplegia, indistinguishable from that due to cerebral hæmorrhage, but the paresis completely clears up in a few hours.

The treatment consists in venesection followed by morphine. The former is sometimes dramatically successful; if not, a lumbar puncture may be done. Intead of venesection an intravenous injection of hypertonic saline solution is sometimes given—fifty cubic centimetres of a 15% solution, repeated if necessary.

Rheumatic Fever.

In the treatment of rheumatic fever, apart from the routine salicylates or aspirin, or "Tolysin" and local treatment, the following guides may be useful.

In the absence of valvulitis the patient is kept in bed for six weeks, flat on the back; then he is made to rest for six weeks and is under observation for two years.

With valvulitis he is kept for three months flat in bed and then, if the temperature is normal, a sedimentation rate test is done. If the result is positive, the patient is kept in bed and the test is performed every month until the response is satisfactory; after this he is kept for six weeks at rest.

Congestive Failure.

The plan which is, or ought to be, universally used now is to get the patient digitalized as soon as possible, if urgent, and to keep him digitalized by a small maintenance dose, probably for the rest of his life. The dried leaf (or digoxin) preparations are best, but any standardized preparation may be used. In working out the dose it is useful to remember that fifteen minims equal one cat unit of the tincture and that this is equivalent to one and a half grains of the powdered leaf and to one two-hundred-and-fiftieth of a grain of Nativelle's digitalin and 0·25 milligramme of digoxin. A guide to the amount necessary to digitalize a patient is to give one cat unit per ten pounds of body weight. If there is urgency, this may be given in twenty-four hours. For example, for an average person, five pills are given every eight hours for three doses. In extreme urgency it may be given intravenously, as digoxin.

Digoxin is a pure glucoside of digitalis which is absorbed and eliminated more rapidly than digitalis. It can be given intravenously or by mouth. The intravenous dose is 0·75 to 1·0 milligramme. After six hours give the drug by mouth. Each tablet contains 0·25 milligramme, and 1·0 to 1·5 milligrammes is an initial dose (this equals four to six tablets), followed by 0·25 milligramme every six hours. Average amounts are 1·5 to 2·0 milligrammes of leaf, or three and a half to five drachms of the tincture.

Digoxin has largely taken the place of strophanthin. The latter must not be given within forty-eight hours of digitalis. The dose is 0·25 to 0·54 milligramme (one two-hundred-and-fortieth to one one-hundred-and-twentieth of a grain), given intravenously. If there is no urgency, the dose of digitalis may be spread out over a week, with a little added to make up for excretion (one to two cat units daily). When digitalization has been accomplished, a maintenance dose of 0·09 gramme (one and a half grains) is given daily or twice daily. This is most important and prevents frequent breakdowns, and many years of useful life may be possible. During digitalization a chart should be kept showing the apex heart rate, pulse deficit, weight, and fluid intake and output. Signs of overdose are anorexia, headache and nausea, and later, vomiting, visual disturbances (especially for colour) and diarrhœa. It is important to remember that nausea and anorexia are also signs of congestive failure, so that during the first day or two of treatment these symptoms should not be regarded as due to digitalis overdose. Coupled beats are a sign of which to beware. Digitalis should be used in congestive failure with regular rhythm, just as with irregular rhythm, though the effect is not so good. Tonic doses, except as maintenance doses after digitalization, are useless.

If the above scheme has failed to give relief, the next step is to use one of the mercurial diuretics, the best being "Salyrgan" and "Mersalyl". "Salyrgan" is best given intravenously, 0·5 cubic centimetre the first day, then 1·0 cubic centimetre, then 2·0 cubic centimetres; and it may be usefully combined with chloride of ammonia, 1·0 to 2·0 grammes (15 to 30 grains) three times a day. If nausea results, the chloride of ammonia is best omitted. The latter dose is then repeated once or twice a week, as indicated, for months or even for years. It is extraordinary how effective this constant dose is. Old œdematous patients, previously without hope, can be got back on their feet and with care remain in fair health for a long time. Should this treatment fail, the drastic treatment of thyreoidectomy should be considered. The cases suitable require careful selection, for the end result may be worse than before; but I have seen some excellent results in apparently hopeless cases, so that one cannot dismiss this question with a shrug.

Total thyreoidectomy is the aim, so that myxœdema results, with much lowered basal metabolic rate—down to 20% or 30% below the normal for the patient. Added to this is the drop due to improvement in the heart condition, which is itself responsible for raising the basal metabolic rate 30% or more. The relief to the heart's work is obvious. The resultant myxœdema may be somewhat uncomfortable for the patient, but very much easier to bear than the distress of waterlogging *et cetera* caused by a failing heart.

BRONCHIECTASIS.[1]

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EVERY year a considerable number of cases of chronic lung disease are seen in the out-patients' department and wards of the Royal Alexandra Hospital for Children. The majority of these are cases of bronchiectasis. It is this children's disease that I shall attempt to describe, omitting the adult type such as may follow tuberculosis, silicosis, cancer *et cetera*. Nor shall I go into the minute pathology of the disease.

Types of Bronchiectasis.

Several types are described.

1. The first type is a congenital variety; it is usually unilateral, and is generalized through the lung involved, affecting in each individual case bronchi of the same size, that is, it is a medium bronchus or a small bronchus affection.

2. In acute respiratory affections, and, above all, in measles, a definite suppurative bronchitis may occur. The bronchial walls become softened, and dilatation, aggravated by coughing, appears. It is seen mainly in small weakly children, and is usually recognized only *post mortem*. Recovery, however,

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 25, 1936.

may occur, and may be clinically complete, as I have seen in a child of three after pertussis, and in one of three cases of diphtheria. Or the inflammation spreads to the interstitial tissues with formation of fibrous tissue. Closely allied to the acute form is that which comes from a subacute or chronic bronchitis, in children the result of continued infections and reinfections from the nose, throat and nasal sinuses.

3. Apart from the congenital type and the very acute form, the essential factor of a bronchiectasis is a preliminary fibrosis of the interstitial tissues, which by its contraction gives rise to the various distortions that occur both in and around the lung itself. We see dilatation of the bronchi, thickening of the lung tissues, pleural thickening and adhesions to the chest wall, pericardium and diaphragm. This fibrosis is initiated in various ways. Inflammation may spread directly from the bronchi to the peribronchial tissues, as seen in nose and throat infections; from an acute or chronic abscess of the lung, which may follow a virulent pneumonia, or the inhalation of a foreign body, or the inhalation of septic material during operations on the nose, mouth and throat; or from infection of a ruptured hydatid cyst; or it may spread from a chronic pleurisy, which is usually the result of an interlobar or pleural empyema. Most commonly the fibrosis begins in a pneumonic area which does not resolve, that is, in the scar of a catarrhal or lobar pneumonia.

The bronchiectatic area is usually in the lower half of the lung. It may be bilateral and diffuse, unilateral or unilobar. When it is bilateral, one side is more affected than the other. Even when bronchiectasis follows a double basal pneumonia, one side only may become bronchiectatic.

We meet with two main types of patient: one is anæmic, thin and under-developed, and in these the "fibrosis" is more apparent than the bronchial dilatation; the other (the later stage) is plump, cheerful and with a high colour, often with a dusky tinge. The former has a dry, irritating, ineffectual cough, a slight evening rise of temperature, poor appetite and a disinclination to play. It is this patient that is usually suspected to be tuberculous, and definite diagnosis is difficult. The latter is a much more "well" child at first sight. There is a rise in temperature only with exacerbations of the inflammation; the fingers are, however, "clubbed", and sputum appears in greater or less amount. In both there are shortness of breath and general weakness. With dilatation of the bronchi and subsequent infection, the cavities become the receptacles for quantities of characteristic grey or grey-brown, fluid, fetid pus. This is discharged at least once in twenty-four hours, but the total amount (100 to 1,000 cubic centimetres) varies, as does the interval between the fits of coughing. Cough is brought on by change of posture, emotion, taking of food *et cetera*. In the disease connected with nasal sinus infection there is a loose phlegmy laryngeal cough, which is very characteristic.

Chest Signs.

Chest signs vary markedly, not only in different cases, but from day to day, or week to week. Again, they may be conspicuous by their absence, even when there is no doubt that definite bronchial dilatation is present. The more marked the fibrosis, the more definite are the signs as a general rule. The signs of cavitation are seldom in evidence. There is deficient expansion, more evident on one side, as well as flattening of the chest wall and diminished vocal fremitus. There may be displacement of the heart towards the affected side, and the diaphragm is raised or does not descend with inspiration. The percussion note is flattened, except over a large and empty cavity. The breath sounds may be weak over the affected area, but usually are definitely harsh, especially at the scapula, with a few crepitations, or a few râles with diminished breath sounds may be heard.

With cavities, the signs depend on the fullness or otherwise of such cavities. All signs are intensified with each fresh attack of inflammation and consolidation, and may practically disappear again as the inflammation subsides. It is at these times of exacerbation that the condition may be first suspected; and many patients are sent to hospitals as sufferers from pneumonia, as indeed they are, with a history of numerous previous attacks of pneumonia, but without a recognition of the underlying bronchiectasis. Even an attack of bronchitis will throw the affected area into relief.

Prognosis.

The disease is not incompatible with a fairly long and comfortable life. In advanced cases the patients die of a general septic condition or cerebral abscess.

Diagnosis.

Diagnosis is quite easy in the marked cases, but it is not always easy to discover the causal factor. When the condition is suspected, X ray examination of the chest, with or without lipiodol, provides the diagnosis. Tuberculosis and asthma have to be excluded, and this may be very difficult.

Treatment.

1. Early recognition of the conditions which predispose to this disease must be our first aim. A foreign body in the lung, nasal infections, nasal sinus troubles which may occur even before the age of five, a small pleural or interlobar empyema—all these must be diagnosed early if we wish to prevent fibrosis. Patients with pneumonia must be followed up until resolution has been complete; iodides, tonics, change of climate *et cetera*, must be ordered; and no empyema should be operated upon while there is still a massive consolidation in the lung. Particular attention should be paid to measles and pertussis.

2. Medical treatment is in the main palliative, but especially in the early and even in the late cases much can be done to minimize the spread of

the disease. First of all, the patient should live in a dry climate, good food should be given, with *Oleum Morrhuæ*, iodides and inhalations containing creosote, menthol *et cetera*. Drainage by posture should be used; and I am a great believer in vaccines. Postural drainage has proved of great help, but the "beneficial posture" depends on the situation of the area to be drained. Usually drainage is very satisfactory with the head down over the edge of the bed, but I have recently seen a very adverse criticism of this procedure.

Lipiodol, used as a diagnostic agent, has, in some of my cases, proved of benefit, but I would scarcely advise it as a routine method of treatment. It does seem to minimize the odour and the amount of sputum.

3. General drainage of the tubes and cavities may be performed with the bronchoscope.

4. As regards the place of surgery in the treatment of this disease, I cannot speak as a surgeon, but only as a physician. When there is a definite dilatation of the bronchi, I cannot see that a temporary compression of the lung can be of any lasting benefit, or can effect a cure. Pneumothorax can help, to my mind, only by giving the infected cavities a chance to become less infective. The cavities will still be there at the end of the treatment, even when complete collapse of the lung is brought about. It is the same with phrenic avulsion, except possibly in those rare cases in which a remote basal cavity is found. The application of direct external drainage of the lung may be possible, in picked cases, to a single large cavity with commencing bronchiectasis around it. Thoracoplasty, to be of use, must obliterate the affected area. There only remains a surgical removal of the affected lung itself.

OTO-RHINO-LARYNGOLOGICAL CONSIDERATIONS IN BRONCHIECTASIS.[1]

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PATIENTS with bronchiectasis present themselves not uncommonly to the oto-rhino-laryngologist. Some come for bronchoscopic examination or treatment, others come for treatment or investigation of known or suspected sinusitis, whilst others attend for lesions quite unconnected with bronchiectasis. The occurrence of bronchiectasis in the practice of this specialty is relatively high, but it is not truly representative of its occurrence in ordinary practice. In general practice the numbers of patients with dry or comparatively dry bronchiectasis, and also with bronchiectasis secondary to acute pulmonary disease, would undoubtedly be relatively higher. From this it follows that we in oto-rhino-laryngological practice see fewer of the type of dry case that some consider needs no treatment, and also fewer of the unilateral type, secondary to acute pulmonary disease, which is suitable for such a radical procedure as lobectomy.

It is not easy to place these intermediate cases in their true perspective. We know that the disease in this class is incurable when once fully developed, yet amongst these are patients who not only look well, but who enjoy very fair health. Statistics of a hopeful or pessimistic outlook can be invoked at will. As an example of the former, one might quote Clarke, Hadley and Chaplin,[1] who followed 45 patients for eleven years with only three deaths. They remark that the health of the majority was excellent. Apart from those that really do enjoy good health are those many more who do not, but who look healthy on account of the increased width of the face caused by chronic antritis. Graham Brown[2] emphasized this, and he termed the appearance "pseudo-robust". On the other hand, Roles and Todd[3] followed 106 patients for three to six years. Of the 49 patients treated medically for only this short time, 23 died and nine others were incapacitated. McNeil[4] has demonstrated that the highest mortality occurs in the first few years after onset.

The oto-rhino-laryngologist, however, does more for potential sufferers from bronchiectasis than all his colleagues put together by curing those conditions which, if left, would in many cases lead on to bronchiectasis. Almost daily he sees patients who have a chronic loose cough and who present themselves in any stage between the recent onset of a sinusitis up to a fully deveolped bronchiectasis with putrid infection, though such putrid cases are admittedly uncommon in children. Some of these cases are as pitiable as any we encounter. As a result of suitable treatment of the upper respiratory tract, the milder of these lower tract infections often clear up, sometimes very rapidly. Such treatment also diminishes the frequency of acute pulmonary diseases, which in turn might produce that unilobar type of bronchiectasis suitable for lobectomy. Also foreign bodies, if not removed, would produce bronchiectasis.

In a short paper there is time only to refer to a few aspects of the subject under consideration, and to deal with only the following headings will be attempted: (i) upper respiratory infections and bronchiectasis, (ii) bronchoscopy and bronchiectasis, (iii) cerebral abscess and bronchiectasis.

Upper Respiratory Infections and Bronchiectasis.

Only in recent years have infections in the nose been accepted as a common cause of chronic bronchitis and its sequelæ.

In 1925 Adam[5] pointed out the connexion between sinusitis and bronchiectasis, and later Chevalier Jackson confirmed it. In this country the recognition of the relationship is largely due to Graham Brown, who wrote on the subject in

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 25, 1936.

1928. It is surprising, however, that in some countries, particularly Great Britain, quite comprehensive papers on bronchiectasis are still published without a reference to sinusitis as a cause.

As late as 1929 so great an authority as Frazer[6] had not grasped the significance of the relationship, for he asked in public whether there was supposed to be any particular connexion between bronchiectasis and accessory sinus disease. He added that unless the association of the two conditions was of frequent occurrence, he doubted whether the connexion with a close one.

The occurrence of sinusitis with bronchiectasis is shown by the following figures. Quin and Meyer[7] found them associated in 57·9% of cases, whilst Clerf[8] in a series of 200 cases found sinusitis in 82·4%, and Kernan[9] states that bronchiectasis almost always has origin in sinusitis. Of the 56 Newcastle General Hospital patients with bronchiectasis who have had the sinuses examined by X rays since 1931, there has been evidence of sinusitis in 50, or 89·3%. Of the 50 with sinusitis, the antra were involved in every instance. In 19 cases, or 38%, the antra were involved alone. In 31 cases, or 62%, other sinuses were also involved.

In Australia it is quite the exception to find bronchiectasis, especially in children, without sinusitis, but I do not believe that all cases are caused by the sinusitis in the first place. It would be strange if sinusitis did not occur when large quantities of infective sputum are expectorated, though Clerf[10] asserts that such upward infection is rare. Many of the unilateral cases of bronchiectasis are secondary to acute pulmonary disease, and I believe that in some of them the sinusitis found co-existing later is due to a spread from the bronchiectasis; but by airing such an opinion I do not wish to be understood to belittle sinusitis as a cause of bronchiectasis. It is definitely the most important cause in childhood. After the two lesions are established, whatever the sequence of infection in the particular case, a bagatelle effect is produced, one lesion reinfecting the other; and, once the sinusitis is present, no treatment of the bronchiectasis is likely to be of any benefit until the sinusitis is under control.

Other lesions of the upper respiratory tract are far less likely than sinusitis to produce bronchitis and the sequelæ leading up to bronchiectasis. Atrophic rhinitis and then extensive pyorrhœa are probably next in order of frequency. Chronic tonsillitis alone is a rare cause. Some years ago I performed saline suction lavage on all antra in children when I removed tonsils and adenoids. I found antral infection in most cases when a loose cough was present. Stress in this investigation is laid on using the prone position, using straight puncture needles which can reach the posterior part of the antra and the use of fluid suction lavage.

In the majority of cases of bronchiectasis with sinusitis a bilateral lung lesion is present, so that when the condition is fully developed the prognosis is correspondingly bad as regards cure. Of the 50 bronchiectasis patients at the Newcastle General Hospital with sinusitis, only four, or 8%, had unilateral bronchiectasis.

The spread of infection from the sinuses to the bronchi is attributed by different schools mainly to spread by lymphatics or by aspiration.

No really satisfactory exposition of lymphatic spread has yet been made, in spite of lymphatic spread having been considered in all seriousness. If downward lymph spread from the sinuses to the tracheal submucosa could be demonstrated, it would be easy to understand the infection's continuing by the anastomosis with the periarterial and peribronchial lymphatics to the lung. But such lymphatic connexions have not been demonstrated.

The other possible method of lymph spread invokes a partial hæmatogenous spread to aid it. It suggests an initial lymphatic spread to the deep cervical glands, and thence by the thoracic and right jugular ducts to the great veins, from which the infected blood carries the infection up the pulmonary artery.

It is much more reasonable to believe in the aspiration theory. Certain experimental evidence supports the possibility of this. Quin and Meyer[7] showed that of 11 patients who had lipiodol instilled when asleep, five inspired some; but the importance of the experiment is somewhat vitiated by the fact that it was necessary to give the patients morphine in order to avoid rousing them. Argyrol drops in the nose can easily be sniffed into the trachea, and can be demonstrated by indirect laryngoscopy.

I believe that it is because infection spreads by aspiration that bronchitis occurs frequently with sinusitis when there is a comparatively copious discharge, but seldom in chronic tonsillitis, in which discharge is scanty. If lymph spread were the commoner, I believe chronic tonsillitis would more frequently produce a lower respiratory lesion than would sinusitis, for cervical lymphadenitis is far more prone to occur in tonsillitis than in sinusitis.

Bronchoscopy and Bronchiectasis.

Endoscopic procedures are of use in bronchiectasis as a means of diagnosis or of treatment.

Lipiodol examination is the means by which a definite diagnosis is made, but in the cases with the more profuse sputum a diagnosis from the history and physical examination is usually not difficult. Priddle,[11] in a series of 51 cases, found that a correct clinical diagnosis could be made in 47 instances without lipiodol. However, it is probable that if the use of lipiodol were not regarded as essential, the dry type of case, of which Wall and Hoyle[12] collected 20 of their own in two years, would be entirely missed. In the lipiodol examination, bronchoscopy is of use in removing the secretion before the injection of iodized oil. Such removal is of advantage in diagnosis when the secretions are very viscid, as it removes one of the causes of poor filling of the bronchial system.

When a diagnosis of bronchial obstruction has been made, bronchoscopy is necessary in order to

determine the cause of the obstruction, and treatment can be given in the form of removal of unsuspected (and often radio-translucent) foreign bodies, dilatation of strictures, or removal of newgrowths *et cetera*. When no bronchial obstruction exists, bronchoscopy is still of use in treatment. Removal of bronchial secretions will often give considerable relief both in diminishing the amount of the secretion and in reducing its fetor. In such cases the secretions removed will often not flow if the glass container in which they have been collected is inverted. In unilateral cases repeated bronchoscopy is of the greatest use in preparing the patient for the operation of lobectomy, and Tudor Edwards[13] stresses this.

Recently an over-optimistic wave of enthusiasm, particularly in America, over-estimated the results of bronchoscopic aspiration; but it is now realized that only the mildest cases can be cured by such means alone.

The amount of discharge in the bronchi in the damper cases of bronchiectasis makes endoscopy difficult. Negus has helped in this respect by having the small Chevalier Jackson light bulbs made with a lens which throws a brilliant beam ahead in the direction of the area to be inspected. This greatly increases the illumination. Scott Pinchin and Morlock[14] have assisted further by several modifications to the Chevalier Jackson bronchoscope. A telescopic system of lenses is substituted for the usual direct vision air channel. Apart from the remarkable optical advantages of this system, the blocking of the viewing channel of the bronchoscope prevents discharge being blown over the examiner when the patient coughs. During the examination the patient breathes alongside the bronchoscope.

Cerebral Abscess and Bronchiectasis.

Bronchiectasis is well known to be the next most common cause of cerebral abscess after *otitis media*. Jex Blake[15] found cerebral abscesses 15 times in 105 *post mortem* examinations in cases of bronchiectasis.

The distance from the lung to the brain is such that metastasis has been invoked as the cause without much thought of any other possible cause. There is, however, another explanation that was first suggested by Adam.[16] He suggested that as sinusitis was so prevalent as to be almost concomitant with bronchiectasis, these cerebral abscesses might be secondary to such sinusitis and not be due to the bronchiectasis.

This is an interesting suggestion, and I think that probably it is the correct one, but an attempt to verify the matter one way or the other has not met with much success.

First, one would expect that a lesion which produced metastatic lesions in the brain would produce metastatic lesions in other organs. Metastatic abscesses do not appear to occur commonly in bronchiectasis, and I think this is the chief reason why Adam is probably correct in his surmise.

Secondly, one would expect metastatic abscesses to be multiple, but Eagleton's[7] statistics show that metastatic abscesses are single in 46%. This figure is sufficiently high to make premises based on multiplicity inconclusive. On the other hand, to surmise that multiple abscesses were metastatic would probably be correct.

Thirdly, one would expect cerebral abscesses from sinusitis to be situated in the frontal lobe, as the commonest causes of frontal cerebral abscesses are frontal or ethmoidal sinusitis and orbital cellulitis. Adam[16] pointed this out, and, as a matter of fact, most cerebral abscesses due to bronchiectasis are in the frontal lobe. Unfortunately, Eagleton[17] states that metastatic abscesses are usually frontal in origin also, so this line of argument also is not conclusive.

Fourthly, metastatic abscesses are said to occur with an apoplectic onset concurrently with the infective infarct. Unfortunately, insufficient details of cases are available for me to say whether such an onset is common or otherwise in bronchiectasis.

Although, therefore, nothing in the way of proof is offered that cerebral abscess in bronchiectasis is usually secondary to the sinusitis that accompanies it, the suggestion is well worth consideration.

Gowers[18] stated that pulmonary metastatic abscesses were three times as common on the left side as on the right.

There is a belief that lobectomy predisposes the patient to the onset of cerebral abscess in bronchiectasis, but evidence at *post mortem* examination after lobectomy may show that the abscess is an old one. Roberts and Nelson[19] reported such a case in which the abscess had a very thick capsule. It is possible that cerebral abscess has been found *post mortem* after lobectomy and has been blamed as the cause of death, when it was quite latent and was not the actual cause. There is no site in the brain where abscesses have such a tendency to be latent without any symptoms whatever as in the frontal lobes. Eagleton[17] states that a large proportion of the cases of frontal lobe abscess were not diagnosed during life, a sudden and fatal termination having occurred as the result of the rupture of the abscess. If, therefore, a lobectomy was performed during the latent period, it is not unlikely that the abscess found, should the patient die, might be considered a complication precipitated by the lobectomy.

Conclusion.

Lack of time available prevents the discussion of more than these three aspects of bronchiectasis, and I regret that it has been impossible to deal even with these more fully; but if, by my emphasis of the importance of the treatment of sinusitis before the milder degrees of bronchitis in children have been allowed to progress on to hopeless bilateral bronchiectasis, I have stimulated any who were previously half-hearted about such measures, I shall feel well rewarded for presenting this paper.

References.

(1) Clarke, Hadley and Chaplin: "Fibroid Diseases of the Lung", 1894; quoted by C. McNeil, "Notes on Bronchiectasis", *The British Medical Journal*, Volume II, August 6, 1932, page 229.
(2) R. Graham Brown: "Bronchiectasis in Children: The Pseudo-Robust Appearance in Cases Associated with Nasal Accessory Sinus Suppuration", *Journal of Laryngology and Otology*, Volume XLIII, September, 1928, Number 9, page 656.
(3) C. Roles and G. S. Todd: "Bronchiectasis: Diagnosis and Prognosis in Relation to Treatment", *The British Medical Journal*, Volume II, October 7, 1933, page 639.
(4) C. McNeil: "Notes on Bronchiectasis", *The British Medical Journal*, Volume II, August 6, 1932, page 229.
(5) J. Adam: "Is Chronic Maxillary Sinusitis a Cause of Bronchiectasis?" *Journal of Laryngology and Otology*, Volume XL, March, 1925, page 172.
(6) J. Frazer, speaking at the thirtieth meeting of the Scottish Society of Otology and Laryngology at Glasgow, November 30, 1929; proceedings reported in *Journal of Laryngology and Otology*, Volume XLV, April, 1930, page 272.
(7) L. Quin and O. Meyer: "Relationship of Sinusitis and Bronchiectasis", *Archives of Oto-Laryngology*, Volume X, Number 2, August, 1929; abstracted in *Journal of Laryngology and Otology*, Volume XLIV, December, 1929, Number 12, page 853.
(8) L. H. Clerf: "Interrelationship of Sinus Disease and Bronchiectasis, with Special Reference to Prognosis", *Laryngoscope*, Volume XLIV, July, 1934, page 568.
(9) J. D. Kernan: "Endoscopy in Treatment of Disease of Upper Respiratory Tract", *Laryngoscope*, Volume XLV, July, 1935, Number 7, page 503.
(10) L. H. Clerf: "Bronchiectasis Associated with Disease of Nasal Accessory Sinuses", *Archives of Oto-Laryngology*, July, 1927; abstracted in "Practical Medical Series Year Book", "Eye, Ear, Nose and Throat", 1927.
(11) W. W. Priddle: "Bronchiectasis: An Analysis of 51 Cases", *New York State Journal of Medicine*, September 15, 1930; abstracted in "Practical Medical Series Year Book", "Eye, Ear, Nose and Throat", 1930.
(12) C. Wall and J. C. Hoyle: "Observations on Dry Bronchiectasis", *The British Medical Journal*, Volume I, April 8, 1933, page 597.
(13) A. Tudor Edwards and C. Price Thomas: "One Stage Lobectomy for Bronchiectasis: An Account of 48 Cases", *The British Journal of Surgery*, Volume XXII, October, 1934, page 310.
(14) A. J. Scott Pinchin and H. V. Morlock: "Bronchoscopic Apparatus", *The Lancet*, March 26, 1932, page 671.
(15) Jex Blake: *The British Medical Journal*, Volume I, May, 1920, page 591; quoted by A. Tudor Edwards and C. Price Thomas, "One Stage Lobectomy for Bronchiectasis: An Account of 48 Cases", *The British Journal of Surgery*, Volume XXII, October, 1934, page 310.
(16) J. Adam: "Note on Connection of Brain Abscess with Bronchiectasis", *Journal of Laryngology and Otology*, Volume XLI, February, 1926, page 93.
(17) W. P. Eagleton: "Brain Abscess", 1922.
(18) Gowers: Quoted by Eagleton, *loco citato*, page 74.
(19) J. E. H. Roberts and H. P. Nelson: "Pulmonary Lobectomy Technique and Report of 10 Cases", *The British Journal of Surgery*, Volume XXI, October, 1933, page 277.

Reports of Cases.

DIABETIC GANGRENE.

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A.R., A MALE, aged seventy-four years, was brought to me from the country suffering from gangrene of the anterior calf muscles and the forepart of the foot of the right leg, with considerable pain below the knee joint posteriorly. His first complaint was of severe pain in the leg, as described. A doctor was consulted, who, in view of a slight rise of temperature and general malaise and an epidemic of influenza prevailing, diagnosed the condition as influenzal. Within a day or two the gangrene appeared and the patient was brought to me. The diagnosis was easy then. Definite arteriosclerosis and urine loaded with sugar proclaimed an emergency. Appropriate treatment was instituted and the urine soon became sugar-free. The gangrene had an unusual distribution. It is of the management of this aspect of the case that prompts me to report it.

Treatment began on August 13, 1935, with wet carbolic compresses (1 to 40). In three days this was changed to free dusting with boric acid covered with boric lint and wool. The severe pain was completely relieved by *Pilula Codeinæ Phosphatis*, 0·06 gramme (one grain), repeated as required. (Codeina is the drug for pain and unrest in diabetes.) The foot, up to the present time, has remained in a state of dry gangrene. The anterior tibial muscles softened and sloughed away. The greater part was cut away to hasten matters and the remainder has organized. The broad external surface of the tibia was bare and necrosed, including the crest. It is now almost reorganized by granulations sprouting from within. The picture of the wound, as shown in Figure I, shows all

FIGURE I.
Showing condition of wound.

but a small area of the bone so covered. The wound is slowly filling by granulation. There has not been a drop of pus at any time. The glistening spots are from clear yellow serum, which lies like a small lake at the bottom of the wound. The wound has never been packed; in fact, care has been taken to keep the dressing from coming in contact with it. After exposure in dressing, the cavity has been gently mopped out with swabs moistened with antiseptic lotions. At first, perchloride of mercury, 1 to 5,000, was used. Later the lotion was changed to "Dettol" and now "Melasol" is used both for the dressing and for mopping. The dressing, always bulky with the superimposed boric wool, is changed daily or on alternate days. The interval could be lengthened if personal supervision were possible.

The general condition of the patient is excellent on a line ration diet. He refused insulin. He is encouraged to move the leg by flexion and to raise and lower it when sitting down or reclining. He gets about well on crutches. Figure II shows the distribution of the gangrene. The

FIGURE II.
Showing distribution of gangrene.

general pallor is due to boric acid. The case shows an unusual distribution of gangrene, the healing of this wound without any suppuration, the reorganization of slough and of necrosed bone under carefully designed antiseptic treat-

ment, the main feature of which is the prevention of the entry of organisms from without, avoidance of irritating the wound by the dressing, and the minimal application of antiseptics therein.

Reviews.

EAR, NOSE AND THROAT DISEASES.

"DISEASES OF THE NOSE, THROAT AND EAR", edited by Logan Turner and under the authorship of several of the most prominent oto-rhino-laryngologists of the Edinburgh school, is now presented in a fourth edition.[1]

Since the late Major Porter produced his little book on diseases of the nose, throat and ear, and throughout the many reprintings, revisings and enlargements under the editorship of Logan Turner, this text has always comprised a masterly and explicit summary of the diseases and disorders in this special department of medicine. The fourth edition, now published, continues to maintain the same qualities, while it includes some account of recent advances in theory and methods. Some changes in authorship have been made in certain sections, but without any gross variation in the general plan. The host of beautiful illustrations and diagrams of past editions has been even further augmented.

That this widely accepted British text will continue to hold pride of place is unquestionable. While the undergraduate medical student has long ceased to have such a small and compact work as Porter originally presented, nevertheless compensation comes by the extension of its sphere of use to the student specialist and to the practitioner who, perhaps, does require a more extensive treatise for his surgical library.

CLINICAL DIAGNOSIS.

THE abrupt change from the academic study of anatomy and physiology to personal contact with sick people necessarily results in a difficult period for the medical student. In order to aid the student at this time, E. Noble Chamberlain has written a book entitled "Symptoms and Signs in Clinical Medicine" as "an introduction to medical diagnosis".[2]

The opening chapter deals with the routine interrogation and examination of the patient, and includes a very useful table of main symptoms. By means of this table the student can ascertain some of the further questions which should be asked, and the systems primarily requiring investigation.

The subsequent chapters deal *seriatim* with the general external characteristics of disease and the various systems: respiratory, cardio-vascular, urinary, digestive, hæmopoietic and nervous.

Symptoms and physical signs are recounted in such a manner that the student can arrive at a conception of the pathological processes causing them. Some of the commoner diseases affecting the respective systems are described, in order to facilitate an appreciation of the correlation of symptoms and physical signs in forming a diagnosis; and at the end of each section appropriate comment is made on the special investigations which may be required in establishing a diagnosis.

Sections dealing with fever, medical operations and instrumental investigations and clinical pathology and biological chemistry are included; and a chapter on the examination of sick children by Norman B. Capon is a valuable addition to the book.

A clear, concise description of clinical methods is given, but brevity has resulted in apparent dogmatism, which detracts to some extent from the value of the work for advanced students and graduates. We do not like the implication that Heberden's nodes are found only in "advanced and usually non-progressive arthritis" and that the "apices are palpated" and "the lower lobes may be examined" by placing the hands on the chest in certain situations.

Heavy percussion is strongly recommended; but the majority of students experience very great difficulty in mastering the art of percussion, and the heavier the percussion, the greater the difficulty of interpretation. Light percussion is more readily practised by, and is infinitely preferable for, the beginner.

In dealing with cardiac diseases the author states that dyspnœa is due to pulmonary congestion, a very insufficient explanation; and the importance of distension of the veins of the neck as a sign of congestive failure is not stressed. Occurrence of the normal third sound is not differentiated from gallop rhythm. The statement that the presystolic murmur of mitral stenosis is part of a prolonged diastolic bruit is incorrect. The disappearance of the presystolic murmur with the onset of auricular fibrillation and Crighton Bramwell's graphic records are evidence of the importance of auricular contraction.

There are other similar criticisms which might be made, but they are all minor points and do not appreciably detract from the real value of the work, which is a most useful publication and well fulfils its purpose. It is well printed, save for occasional dropped letters, and profusely illustrated.

"AN APPLE A DAY."

BOTH the authorship and the title of the latest book on individual hygiene should attract the interest of the still comparatively small number of the public who realize that the preservation of health is a personal responsibility rather than a combination of heredity and chance.

"An Apple a Day", by Sir William Arbuthnot Lane, is written along the usual lines.[1] It is divided into two parts. In Part I the importance of prenatal forethought by the parents, of diet in infancy and childhood, of clothing, and of the values of the various vitamins are laid down in accord with modern orthodox views. Some very sound advice is given with regard to the nervous system, on the importance of mental hygiene in childhood and the lasting effects of discord in the family life, of frustrations, conflicts and undue emotionalism. The interdependence of the endocrine and nervous systems is also pointed out. There are a few words on growing old.

Part II deals with "Some Common Ailments". Constipation (as we know) the author regards as "the disease of diseases". "Bilious attacks" are not explained with any great degree of exactitude. When "nasal colds and catarrh" are prevalent, "unless you have something to say or to swallow, the mouth should be kept shut". (Deglutition with the mouth open must be rather a feat). Rheumatism, neuritis, headaches, eye strain, diseased tonsils and adenoids, asthma and bronchitis are discussed in a more or less informative but not very useful way on account of the lack of available space.

Those who digest and assimilate "An Apple a Day" will not only "keep the doctor away", but may also enjoy happier and healthier lives. The trouble is that it may be read casually, imperfectly digested and very little absorbed.

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[1] "Diseases of the Nose, Throat and Ear for Practitioners and Students", edited by A. Logan Turner, M.D., LL.D., F.R.C.S.E.; Fourth Edition, revised and enlarged; 1936. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 491, with 243 illustrations in the text and 21 plates. Price: 20s. net.

[2] "Symptoms and Signs in Clinical Medicine: An Introduction to Medical Diagnosis", by E. N. Chamberlain, M.D., M.Sc., M.R.C.P., with a chapter on the Examination of Sick Children by N. B. Capon, M.D., F.R.C.P.; 1936. Bristol: John Wright and Sons Limited. Royal 8vo, pp. 435, with 282 illustrations, of which 17 are in colour. Price: 25s. net.

[1] "An Apple a Day", by Sir W. Arbuthnot Lane, Bt., C.B.; 1935. London: Methuen and Company, Limited. Crown 8vo, pp. 184. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, JULY 25, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

INTESTINAL OBSTRUCTION.

WHEN medical practitioners speak of intestinal obstruction they refer to a condition in which the lumen of the bowel is occluded, but they generally think also of intestinal strangulation in which circulatory involvement overshadows the mere blocking of the intestine. Every medical practitioner who has to deal with what is known colloquially as the "urgent abdomen" has learned to recognize the shock and collapse caused by a volvulus, an intussusception or strangulation by a band. He knows that unless operation is performed without delay his patient has little hope of recovery. The blocking of the lumen of the gut by a new-growth, by a gall-stone or by a kink will eventually lead to the same fatal issue; but, if the blocking has not occurred high in the intestinal tract—in the duodenum or the jejunum—the patient's condition may be good and may remain good for some days in spite of abdominal pain and distension. Operation, of course, is none the less urgently indicated. In the introduction to their report on an experimental study of intestinal strangulation,[1] G. C. Knight and D. Slome, Leverhulme Research Scholars of the Research Department of the Royal College of Surgeons of England, draw attention to the high mortality rate that is still associated with acute intestinal obstruction. They hold that it cannot be too strongly insisted that the term acute intestinal obstruction is only a general one, covering a variety of pathological conditions, in some of which at least the obstruction *per se* is the least important factor. They believe that clinical, experimental and statistical findings should be correlated with a definite type of pathological lesion and that the results obtained in the investigation of one group should be applied to that group only and not to the subject as a whole. If this were done, the pathogenesis of the several forms of intestinal obstruction would become clearer and unexpected advances in treatment might become possible. Knight and Slome point out that in acute intestinal strangulation the essential pathological feature is venous strangulation of the intestine, and their investigations have been confined to this aspect of the subject. Most of the experimental work so far carried out has been devoted to intestinal obstruction without strangulation.

Before describing their own investigations Knight and Slome discuss the effects that obstruction alone may produce; and they conclude that the main evidence goes to show that death from simple obstruction is due to a toxæmia which is the result of protein degradation occurring either in the bowel lumen or in the wall of the obstructed segment. In their early work they were able to confirm the findings of other observers on the difference in the length of survival time after simple obstruction, after total anæmia or obliteration of the circulation and after venous strangulation. The survival time after venous strangulation is short and its physiological consequences are set out; they fall into two groups, one having to do with loss of fluid from the circulation and the other with toxæmia. Knight and Slome describe their experiments dealing with these two considerations. Their experiments on the loss of fluid from the circulation were ingeniously planned, and they appear to be justified in their conclusion that fluid loss plays no more

[1] *The British Journal of Surgery*, April, 1936.

than an accessory rôle in the production of the circulatory collapse of intestinal strangulation. Two possibilities remain: a chemical and/or a nervous factor. Knight and Slome have deliberately deferred consideration of a nervous factor, and in this respect their work is incomplete. They prove, however, that a toxic factor does operate, and we are left to speculate on the possible participation of the nervous system in the fatal result. A toxic depressor substance appeared in the peritoneal fluid of their laboratory animals, sometimes within twelve hours of strangulation; the time of appearance of the depressor substance was governed by the severity of the strangulation. The depressor substance was also found in the blood of the gut. Experimental evidence is produced for the conclusion that the depressor substance arises in the wall of the gut itself and passes from there both into the lumen and into the venous blood. Since the substance is not present in the lumen before strangulation, and since the rapidity of its appearance in high concentration appears to exclude a bacterial origin, the toxic substance is held to arise from the tissues of the gut wall as the result of intrinsic changes consequent upon venous strangulation. Proof that the depressor substance passes into the veins is offered by the observation that when the venous obstruction was relieved and the blood was allowed to return to the circulation, a further fall in the animal's blood pressure always occurred.

In this account of the work of Knight and Slome several important side issues have not been mentioned. The main conclusions that have been stated, however, raise some interesting questions in surgical practice. We may ask, for example, whether a patient who is sorely stricken with an acute strangulation will be able to withstand the flooding of his circulation with additional toxic substance when the strangulation is relieved. We know that very often he will not; and sometimes, of course, he has received a lethal dose of toxic substance before he comes to operation. Knight and Slome would have statistical studies undertaken in comparable cases of equal severity to contrast the results of resection with those of relief of the strangulation. This would be very difficult to do. Even if such a statistical study showed that one course of action was more likely to succeed than another, the procedure actually adopted would depend on the experience and judgement of the surgeon. Perhaps future work will reveal a method of counteracting the effects of the toxic substance. If this does happen, it will be a boon to surgery and will save many lives; but, even so, the safety of the patient will really depend, as it does now, on accurate diagnosis and immediate operation.

Current Comment.

HODGKIN'S DISEASE OF BONE.

IN Hodgkin's disease attention has been so closely focused upon the glandular implication that other manifestations have tended to be overlooked. In 1929, however, W. P. Blount reported, with X ray illustrations, a case in which involvement of the spine and shoulder preceded general enlargement of the lymph glands by two years. In this case diagnostic errors were made. Blount quoted the opinion of others that bone involvement was frequent in Hodgkin's disease. Since then other observers have recorded cases of Hodgkin's disease of the skeleton or of the bone marrow and spleen without apparent involvement of the lymph glands.

M. C. Morrison deals extensively with Hodgkin's disease of bone.[1] He comments on the possible primary nature of the bone lesions, favourite sites for which are the skull, spine, sternum and pelvis. Less often are the ribs, clavicle, humerus and femur involved. Morrison states that no special symptoms appear to point to bone involvement, although pain due to necrosis, pressure on nerves or invasion of nerve roots may be expected in half of the cases. Signs of cord compression and girdle pains may be observed in vertebral lesions. Spontaneous fractures have been noted. The bone lesions are of four types. There may be pressure erosion from enlarged lymph nodes and due to mechanical interference with blood circulation in the periosteum. This is observed in the dorsal spine. There may be granulomatous periostitis following rupture of the capsule of an adjacent lymph node, as seen in sternal and rib involvement. Or the medulla may be invaded and the marrow replaced by tissue identical in composition with that of Hodgkin's lymph nodes, the result being loss of bone density and disintegration of bone structure. This type is usually associated with boring pain unrelieved by rest. The fourth type shows hyperplasia with

[1] *The Canadian Medical Association Journal*, April, 1936.

increase of the cells normally manufactured there, increased density resulting. Observers have found radiographic changes in bone without clinical evidence of such involvement, and evidence of the disease has been demonstrated at autopsy without radiographic changes being demonstrable. Morrison considers that in the differential diagnosis metastatic carcinoma bears the closest resemblance, especially to the purely osteolytic types, which may be indistinguishable by X ray examination. In lesions with some osteoplasia, differentiation must be made from malignant disease secondary to the breast, thyreoid, kidney or prostate involvement. Periosteal types exclude carcinoma, but syphilis and Ewing's tumour must also be negatived. In infants leuchæmic involvement of bone may be displayed by periosteal thickening due to lymphatic proliferation beneath the periosteum and replacement of the bone marrow by similar tissue. Osseous changes due to lymphosarcoma also may be manifested by irregular condensation with pseudo-cystic areas or erosion and very little new bone formation. A chronic inflammatory process must be excluded, and erosion of dorsal vertebræ by aneurysm may resemble that due to enlarged Hodgkin's lymph glands.

Considering the bone manifestations of Hodgkin's disease with regard to the infectious and neoplastic theories as to its nature, Morrison admits that both in such disease and the various forms of chronic osteitis and infectious granuloma we see areas of destruction surrounded by sclerosis and, except in tuberculosis, new bone formation. The predominant incidence of bone infections in childhood, the acute onset with pain and fever, the tendency to abscess formation, sequestration and frequent periosteal and epiphyseal involvement are characteristic of bone infection. In Hodgkin's disease the intervertebral disk remains intact; in an inflammatory lesion it is usually the seat of early fragmentation and destruction. In Hodgkin's disease the distribution of bone lesions accords with that of metastatic carcinoma. The occurrence of pathological fractures is common to both. Metastasis in bone sarcoma is usually by the blood stream to the lungs, and lymphatic involvement associated with the bone lesions is more in keeping with an inflammatory disorder.

Morrison considers that the evidence tends to support the neoplasm theory. He states that a neoplasm may be defined as a cell proliferation which serves no useful purpose and which does not represent a defence reaction to a foreign agent. The commonly recognized forms are essentially lesions of a local group of cells for an initial part of their course; but in lymphosarcoma the leuchæmias and the Hodgkin's group the frequent involvement of the liver, spleen, lymph glands and bone marrow warrants consideration of an hypothesis of neoplasm of a whole system of cells, such as those of the reticulo-endothelial system; any area in which such tissue is found (including the bone marrow) may be the primary focus. Thus can be reconciled the seeming exception to the rule of primary bone malignant disease being a single lesion. Morrison considers that the explanation of Hodgkin's disease may well be that of the whole problem of cancer. From this point of view his paper is most interesting.

INSULIN PROTAMINE COMPOUND.

In April of this year attention was drawn in these pages to work carried out by Hagedorn and others at Copenhagen on an insulin compound that was sparingly soluble. It was pointed out that this compound, protamine insulinate, is injected as a suspension and that the deposit in the subcutaneous tissue consists of a fluid of practically constant insulin concentration and a steadily diminishing amount of solid particles. The result is that a distinct prolongation of the time of absorption takes place. Hagedorn and his co-workers, as well as Root and others working in Joslin's clinic at Boston, have been insistent that this work is only in the experimental stage. In these circumstances, and since the new preparation will no doubt soon be available in the open market, attention should be drawn to a communication from the Mayo Clinic by R. G. Sprague and others, which confirms the observations already published.[1] Sprague's series comprises twenty patients, nearly all of whom had been the subjects of prolonged observations in hospital "with trials of various schedules of administration of insulin". Ten of the twenty patients represented cases in which control of glycosuria and attacks of acidosis was exceedingly difficult or impossible with the regular water-soluble insulin hydrochloride (insulin-R). Seven other patients had diabetes of such severity that three or four injections daily and a total dose of more than fifty units of insulin-R had been required to maintain control. The remaining patients required from 30 to 50 units daily in two or three divided doses. Ten of the patients were children. The duration of the disease in every case was more than three years and in many cases the patient had been under treatment at the Mayo Clinic for ten years or more.

In most of the twenty cases it was found that with a single administration of insulin-P a better control of glycosuria was attained, as well as a more stable level of the blood sugar, than had previously been possible with multiple doses of insulin-R. Sometimes insulin-P and insulin-R were given together. Emphasis is laid on the observation that although single large doses of insulin-P are apparently safe in severe cases, it is very important that the patient should not refuse his food. It was found that when insulin-P was given alone as a single dose before breakfast, the meals of the first few days provoked glycosuria. When the dose was properly adjusted, however, the level of the blood sugar on successive mornings decreased progres-

[1] *The Journal of the American Medical Association*, May 16, 1936.

sively and the elevating effect of meals diminished, until by the end of from four to six days a normal level of blood sugar might be attained, even in the severest diabetes. When insulin-P was supplemented with small doses of insulin-R the period of obtaining control was shortened. It will be remembered that Root and his co-workers advised that insulin-P should be given in the evenings and insulin-R in the mornings. Sprague and his fellow workers insist, as the other writers have insisted, that insulin-R should be used when quick action is desirable. Their general conclusion is that, although insulin-P in many cases makes effective management of diabetes possible with only one administration of insulin a day and with less insistence on rigid control of the diet, its careless use or disregard of the diet is attended with danger.

ICHTHYOSIS.

ICHTHYOSIS may be of varying grades of severity, the mildest being termed xerodermia. *Ichthyosis simplex* represents a more severe degree, and *ichthyosis hystrix*, or porcupine skin, a more specialized form. The disease is hereditary and congenital, but may not be apparent until some weeks or months after birth. It sometimes runs in families. No sex incidence has been noted. The ætiology of the condition is unknown. A closely allied integumentary state has been observed in *ulcus tropicus*. It has been suggested that it is connected in some way with deficient thyreoid activity, and ichthyotic patients are prone to exhibit a low basal metabolic rate. However, a case on record, of an infant with ichthyosis whose mother had hyperthyreoidism, is not readily explained.

L. M. Mullen states that the disease, although congenital, is not often manifested before the first or second year of life.[1] If it is present in fœtal life to a severe degree, the child is usually stillborn or dies a few days after birth. Mullen accepts the opinion that the condition is the result of a developmental and nutritional defect of the skin. In advanced cases histological investigation reveals dilated vessels, a thickened cutis and the connective tissue condensed into bands. The hair follicles are lengthened and contain lanugo. The glands are generally dilated and cystic. Subcutaneous fat is diminished and sweat and sebaceous glands may both be absent. Prognosis as to cure is unfavourable and the disorder is generally worse in winter than in summer. Cases of ichthyosis have been reported related to idiocy in seven families. Mullen gives details of an instance of ichthyosis in an underweight male infant whose skin at birth resembled cellophane. On the second day after birth the skin became dryer with cracks and raw fissures. By the fourth day flakes of skin were coming off, leaving similar dry skin beneath. These areas came away a few days later, and so on throughout the child's life. The child did not exhibit the usual growth or development and very little weight was gained. Fissures and raw surfaces were practically absent after the first month. No infection developed. The child died at the age of three months. Some skin removed after death showed a very thick layer of keratinized substance on the surface. It was a hyperkeratotic skin, but exhibited no other abnormality.

The family history was interesting. The parents were seemingly normal, but the father's sister was said to have had a "delicate skin". The first child of the union, a male, apparently healthy, was found dead in bed at the age of six months. The second child, a male, and the fourth, a female, were living at the time of the report, aged five and two years respectively. The third child, a male, died at the age of one month. It had a congenital umbilical hernia and a skin similar to that of the fifth child, which was the patient reported. Mullen could not find in the literature any mention of other congenital abnormalities associated with ichthyosis. One can only conclude that the connexion was fortuitous. The literature on ichthyosis is not voluminous, and every case reported would help in elucidating this curious condition. Particularly it might be noted whether there was undue frequency of consanguineous marriages in the parents, as has been observed in the very fatal condition of *xerodermia pigmentosa*.

[1] *The Canadian Medical Association Journal*, April, 1936.

CAUSES OF DEATH IN DIPHTHERIA.

ON the subject of diphtheria Archibald L. Hoyne[1] writes as one having authority, for he has long been the medical superintendent of the Municipal Contagious Disease Hospital of Chicago. Death from diphtheria, he states, may be due to asphyxia, bronchopneumonia or myocarditis. In the first case the diphtheritic membrane forms in or below the larynx and is rarely seen above it; very often, indeed, the presence of membrane in the larynx follows a spread upwards from regions below. Here toxæmia does not kill, but death by asphyxiation follows the physical plugging up of the airway. The complications due to toxæmia—myocarditis and nephritis or various paralyses—are here not found, and no dose of antitoxin, however large, will benefit the patient. Tracheotomy, deftly done, is the only hope.

Nobody can prevent the onset of bronchopneumonia in a case of diphtheria, and it sometimes kills the patient when tracheotomy has already saved his life. Cleanliness in handling the patient is of first importance, and the raising of the foot of the bed, as well as the use of suction apparatus, is of value in lessening the risks of pulmonary complications.

Myocarditis is as a general rule a much later complication to appear than bronchopneumonia. In

[1] *The American Journal of the Medical Sciences*, February, 1936.

severe cases of pharyngeal and naso-pharyngeal diphtheria toxæmia of high degree may appear within three days of the onset unless the antitoxin dosage is sufficient. After the third day the mortality rate rises quickly and steeply. The patient profoundly affected by toxæmia is often curiously alert and commonly suffers from the form of cervical adenitis described as "bull neck". The swelling is soft, suggesting the presence of œdema. In these severe cases the presence of large amounts of albumin in the urine or of an enlarged, tender liver is of the gravest prognostic import.

Long experience has enabled Hoyne to predict with uncanny accuracy the course of events in severe diphtheritic infections. In a typical instance the patient may seem surprisingly well two or three days after the injection of antitoxin; the temperature and pulse rate fall, the exudate disappears and the heart seems undamaged. These appearances are deceitful. Somewhere between the sixth and eighth day the pulse suddenly falls off by thirty to forty beats in the minute. If vomiting and abdominal pain should ensue during the following twenty-four hours, death is inevitable. At necropsy the diagnosis of acute myocarditis is confirmed.

For a considerable time Hoyne and his fellow worker Welford habitually used solutions of glucose in the treatment of diphtheritic myocarditis when once diagnosed. Nowadays they do not wait for the diagnosis, but administer glucose to all patients who have been ill more than three days or who have "bull necks", albuminous urine or a severe post-nasal infection. The glucose is given in the form of a 10% solution in distilled water. This is administered parenterally in quantities up to one thousand cubic centimetres, a dose which is frequently repeated daily for twelve days. In 83 of these serious cases so treated by Hoyne there were ten deaths, a mortality rate of 12%. This figure is in happy contrast to the death rate of from 30% to 60% obtaining in similar cases not treated with glucose. Of the 83 patients, one died within one hour and within twenty-four hours of admission to hospital; and the remainder may be said without exaggeration to have been in a desperate condition. The principle underlying the use of glucose is that diphtheria is held to cause wholesale disturbance of carbohydrate metabolism. The problem of hepatic, and perhaps of renal, insufficiency has to be countered. This should be done before any sign of myocarditis has become manifest. Hoyne and Welford stated over two years ago that cardiac stimulants such as adrenaline and caffeine were of no value in diphtheria.

It is pertinent to note that two workers, Mitman and Begg, have recently reported the successful treatment of diphtheritic paralysis of the diaphragm with the Drinker respiratory apparatus. In one instance normal respiration was resumed after the device had been in operation for four days, and in a second case after fifteen days.

The inevitable reflection arising from the consideration of recent work upon diphtheria is that only folly, inertia and ignorance postpone the day when active immunization will make diphtheria a horrible memory and nothing more. That day is probably far distant. Vaccination was invented 139 years ago, but people still die of smallpox.

A STUDY OF THE ŒSOPHAGUS.

CARDIOLOGISTS and radiologists will welcome the publication of a study of the course of the œsophagus by William Evans.[1] His study is concerned with the œsophagus in healthy subjects and in those suffering from cardio-vascular disease. First of all he studied the œsophagus in three cadavers and then he made radiological observations on various persons after they had been given an opaque meal suspension. He found that citobaryum and a marmalade-flavoured suspension gave the best results. When the œsophagus was filled with the barium suspension and examined radiologically four segments could be identified at which its downward course was interrupted. These took the form of both abrupt and gradual curves; since each is the result of pressure by adjacent viscera, they are referred to as impressions. Evans describes these as the aortic arch impression, the left bronchus impression, the left auricle impression and the descending aorta impression. He found that the left ventricle sometimes made contact with the œsophagus, but that the resulting impression might be absent even when considerable distension of the ventricle was present. It is impossible in the space available to give an account of the normal impressions found by Evans; a general statement of some of his findings in abnormal conditions, however, will, it is hoped, lead radiologists and physicians to study his report in detail—it is illustrated by excellently reproduced skiagrams and diagrams. We find that disease of the aorta or adjacent structures may change the contour of the aortic arch impression. It is elongated and often displaced by syphilitic aortitis and carcinomatous involvement of the bronchial glands. Other conditions above and below the impression cause pulsion or traction of the œsophagus. The aortic impression is also changed by aortic atheroma. The left bronchus impression has been held to be caused by the pulmonary artery; Evans believes that any influence of the artery in causing the impression is indirect and that the left bronchus is directly responsible. The conditions that alter this impression include mitral stenosis and carcinomatous bronchial glands. The left auricle impression may sometimes be altered in mitral stenosis; it is affected by aneurysm of the upper portion of the descending aorta, by hydrothorax, pneumothorax, thoracic neoplasm and fibrosis of the lung. The descending aorta impression is affected by aneurysm and by syphilitic lesions.

[1] "The Course of the Œsophagus in Health and in Disease of the Heart and Great Vessels", by William Evans, Medical Research Council of the Privy Council, Special Report Series, Number 208; pp. 93, with illustrations. Price: 2s. 6d. net.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Dextrose Content of Lumbar and Cisternal Spinal Fluid.

ABRAHAM LEVINSON AND DAVID J. COHN (*American Journal of Diseases of Children*, January, 1936) have studied the comparative dextrose content of the lumbar and cisternal cerebro-spinal fluid obtained from non-meningitic and meningitic patients. The lumbar and cisternal fluid was obtained simultaneously from the same patient, and in the series there were 19 non-meningitic and 38 meningitic patients. The latter included patients with tuberculous meningitis, meningococcal meningitis before and after serum treatment, and meningitis due to the streptococcus, the pneumococcus and the influenza bacillus. In non-meningitic patients the dextrose content of the cisternal fluid was found to be either equal to that of the lumbar fluid or slightly higher. In patients with tuberculous meningitis and in untreated patients with meningococcal meningitis the dextrose content of the lumbar fluid was always reduced. The dextrose content of the cisternal fluid, while usually lower than normal, was not so low as that of the lumbar fluid, and in some instances it was within the normal range. In patients with meningococcal meningitis after serum treatment the values of dextrose in both fluids were much closer to normal. The authors conclude that determinations of the dextrose content of the lumbar fluid are more reliable for the diagnosis of meningitis than determinations made on the cisternal fluid, because the dextrose content of the cisternal fluid is seldom reduced as greatly as that of the lumbar fluid.

Insulin in Malnourished Children.

SAMUEL L. ELLENBERG (*Archives of Pediatrics*, December, 1935) has studied the effects of insulin on ten malnutrition patients at the Lincoln Hospital. An analysis of the cases indicates that if the usefulness of insulin were judged solely on its ability of adding weight to the individual, it is not a very dependable therapeutic agent in malnutrition. In some instances the gains in weight were quite satisfactory, while in others the gain was no more than might be expected had insulin not been employed. In this respect the results agree with those obtained by other observers who concluded that there was no positive evidence that insulin *per se*, when administered with carbohydrate to malnourished infants, produced any appreciable gain in weight. The authors claim, however, that the children receiving insulin showed in a very short time an improved body tone, a better colour, a keener interest in their surroundings, and seemed more active and had more vigour than they manifested before receiving the insulin. The appetite was also considerably improved, although the gain in weight did not necessarily run parallel to the increased appetite. The authors conclude from their study that insulin deserves a therapeutic trial in malnourished children when a general improvement is desired, and that disappointment should not be felt when this improvement is not reflected in a striking gain in weight. They also consider that insulin therapy is worthy of use in the convalescent period following a particularly debilitating and wasting illness.

Pink Disease.

J. VERNON BRAITHWAITE (*Archives of Disease in Childhood*, April, 1936) brings further evidence to bear in support of his suggestion in a previous communication that pink disease is an abnormal reaction to daylight in an infected child. In 1933 the author drew attention to the fact that pink disease usually occurred after an acute infection of the respiratory tract. There is, he maintained, a seasonal incidence in the spring and summer. Protection of the children from daylight by keeping them in a room with windows of ruby glass caused a rapid improvement in their condition. The following additional points are stressed in support of the author's thesis. One patient showed toxic symptoms on being placed in the sunlight, while another was accidentally exposed to sunlight through glass and died on the same day. In the hot summer of 1933 six out of ten patients died, in spite of their being protected from the rays of the ultra-violet end of the spectrum. In 1934 twelve patients were treated by cooling measures—hydrotherapy and light clothing. Only one case terminated fatally, death occurring from ward infection five days after admission. Blood in glass tubes exposed to sunlight was hæmolyzed. This was proved to be due to heat rays. Blood hæmolyzed by heat was shown to be highly toxic to a patient with pink disease when injected subcutaneously.

Epituberculosis.

M. DE BRUIN (*Archives of Disease in Childhood*, April, 1936) discusses the condition of epituberculosis. This term was applied in 1921 by Eliasberg and Neuland to the extensive physical changes occurring at times in the chest in young children who gave a positive reaction to the tuberculin test. The changes, which were mostly localized in the upper lobe of the lung, especially on the right side, completely disappeared after remaining unaltered for some months, the general condition of the patients being comparatively good during this time. Eliasberg and Neuland considered that these pathological alterations were a reaction in the adjacent lung tissue to toxins produced in a tuberculous focus, comparable with the perifocal inflammation which might occur in the proximity of any inflammatory focus. The author, in a series of case records with X ray photographs, stresses the part played by atelectasis in the production of the epituberculous infiltration. In a previous paper he has drawn attention to the occurrence of occlusion of a main bronchus during chronic lung disease. In tuberculosis in particular, which is characterized by swollen glands in the hilus of the lung, it is obvious that pressure of these glands on a main bronchus may occur with consequent atelectasis. Further, on close examination of the skiagrams in some publications on epituberculosis, displacement of the mediastinum to the involved side is found. The author concludes that, although the clinical and X ray findings in epituberculosis are often due to atelectasis, in some instances the infiltration consists of specific tuberculous tissue, resolving in course of time, whilst in others the pathological changes should be considered as a perifocal reaction around a tuberculous process.

The Mantoux Test in Children.

G. GREGORY KAYNE (*British Journal of Children's Diseases*, January-March, 1936) draws attention to the inadequate or inaccurately applied knowledge of tuberculin tests and presents a well-defined routine in both technique and interpretation of the Mantoux test. The author recommends beginning with an intradermal injection of 0·1 cubic centimetre of a dilution of 1 in 10,000 tuberculin and reading the result at forty-eight or seventy-two hours after the test. Ten millimetres of erythema and swelling are necessary to call a reaction positive. If the first test gives no reaction a dilution of 1 in 1,000 is used, and if this gives no reaction the test is concluded by the use of a dilution of 1 in 100. A positive result to a Mantoux test in a child under two years should be regarded as indicating the presence of an active tuberculous process, unless the contrary is proved by further investigation. In a child over two years of age a positive result to a test cannot be regarded as necessarily indicating active disease; but the younger the child, the more likely is the lesion to be active. In older children a positive result to a test merely indicates that tuberculous infection has occurred some time or other in the past. Failure to react to the Mantoux test may be taken to exclude tuberculous infection in general clinical work. Similarly, in the presence of a definite lesion considered to be tuberculous, absence of reaction should lead to a revision of the diagnosis. Finally, the author stresses the importance of the

Mantoux test in the examination of home contacts. It must now be generally realized that a perfunctory history and a physical examination with an occasional X ray photograph cannot be expected to achieve the best results in examination of contacts.

ORTHOPÆDIC SURGERY.

Growth Arrest in Long Bones after Fractures Involving the Epiphysis.

E. L. COMPERE (*The Journal of the American Medical Association*, December 28, 1935) reports a study of all the fractures of the long bones that had been treated in the University of Chicago clinics from October, 1927, to May, 1935; he has tried to determine as nearly as possible the incidence of fractures with growth arrest. The results are shown in a table. Patients treated for fractures of the long bones numbered 693, with a total number of 819 fractures; 211, or 34% of the total number of fractures studied, were in children fourteen years of age or younger at the time the fracture occurred. In this group of children were 290, or 35% of the total number of fractures. In 37 of the 211 cases the fracture involved the growth cartilage. In this group of 37 patients there were 42 such fractures, an incidence of 14·4% of all the fractures in children. In children the total number of fractures that involved the growth cartilage was 42. Of the entire group of 42 fractures, five were complicated by infection and in all of these growth was arrested. There were 33 fractures in which the epiphyseal cartilage was involved, but they were too recent to show deformity at the time of the child's first admission to hospital. The author states that Hans Selye has shown that if the distal end of the femur of a rat is removed during the first few weeks of life, a new growth cartilage is formed and the growth in length is resumed. This new growth cartilage forms always in a plane at right angles to the shaft, irrespective of the plane of the amputation. Fractures of the epiphyseal cartilage of the long bones do not necessarily cause immediate arrest of growth. Growth disturbances may not be apparent six months or even a year after the fracture, but the epiphysis may be expected to fuse earlier than on the uninjured side. Since it has been shown that growth may continue for from one to three years after a birth fracture before the premature closure of the epiphyseal line occurs, in only a relatively small percentage of the cases in which growth is arrested by such fractures will there be an appreciable deformity. When the fracture occurs after the age of twelve years, even though growth may be arrested, a minimal degree of deformity may be expected to result. Haas has stated that trauma is the most frequent cause of disturbances of growth in bones. He further states that the greatest growth activity is localized in the cartilage columns on the metaphyseal side of the epiphyseal cartilage plate, and that, after the destruction of this portion of the growth cartilage, length growth practically terminates. In a series of experiments on young dogs and cats in which Haas damaged the growth cartilage itself, the epiphysis or the metaphysis in various ways, he found that injury to the epiphysis alone or to the metaphysis alone caused very little change in the rate or extent of the growth of the extremity. On the other hand, injuries in the shaft of the bone that extended to and across the epiphyseal line, injuries to the epiphysis that included in the applied trauma a portion of the cartilage plate, or direct insult to the growth cartilage alone did cause an early closure or a complete arrest of growth at the traumatized end of the bone. The author further states that Gatewood and Mullen have also shown that immediate closure of the epiphyses resulted from the destruction of the epiphyseal cartilage plate on the side nearest the joint. Interruption of the extrinsic blood supply to the epiphysis, by stripping away the periosteum at the site of the epiphyseal cartilage caused this epiphysis to fuse to the shaft earlier than in the control limb, although there was no immediate arrest of growth. Haas demonstrated in experimental work that the mere passage of sutures through the actively growing columns of cartilage cells of the cartilage plate resulted in the arrest of growth. The author considers that growth disturbance from fractures near the ends of the long bones in children are more common than is generally recognized and that the clinician should be reserved in his prognosis in such fractures and should follow these patients with periodic X ray examination for from one to two years after union of the fractures has occurred.

Ununited Fracture of the Neck of the Femur.

T. B. MACMURRAY (*The Journal of Bone and Joint Surgery*, April, 1936) discusses the various methods of treating ununited fracture of the neck of the femur and mentions the inevitable unstable hip which follows many of them. In his opinion, Whitman's is the only method which has been practised for a period sufficiently long to enable the majority of surgeons to understand its limitations. The instability of the hip joint may be overcome by one of three methods: arthrodesis, reconstruction as suggested by Whitman and Brackett, or bifurcation, in which the shaft of the femur is transferred directly under the lower margin of the acetabulum and head of the femur. The author has practised this procedure in twenty-seven cases during the last ten years. He emphasizes the necessity of performing the osteotomy at the correct level, otherwise the operation will be a failure. This level should be the lower border of the femoral head, and the osteotomy should be performed in a slightly oblique direction in order that the distal fragment may be pushed inwards and become united with the proximal fragment. The operation may be performed in spite of a very long interval of non-union and it almost invariably gives relief.

Quiescent Tumour Albus and Tuberculous Pseudarthrosis in Children.

A. DELAHAYE (*The Journal of Bone and Joint Surgery*, January, 1936) considers that there are available several different methods of inducing fusion in the slowly developing forms of disease in the knee, such as the unstable, painful, movable knee joint. Intraarticular arthrodesis by transepiphyseal bone-pegging, as simplified by Richard, has often been employed by him in older children. After removal of the articular cartilages a rigid graft, taken from the tibia of the healthy limb, is driven obliquely through the epiphyses. The results obtained by this very simple method are good. Juxta-epiphyseal arthrodesis by means of peripheral grafts, after the manner of Dupuy de Frenelle, also gives good results. Both of these methods are open to the objection that it is necessary to open the joint through tissues in which the disease is by no means extinct. He does not believe that the ultimate growth of the limb is liable to be arrested. In order to avoid any possible trouble from this source, he prefers at present to perform the operation only on children in whom epiphyseal growth and ossification are relatively advanced. Zanoli employs a very similar method with success. He uses a long pedicled tibial graft, which he bends backward and attaches to the femur. The two methods can be employed in combination. With or without modification the operation is of value if used in well-selected cases, and it insures good consolidation. Abundant proof of the solidity of the knee has been forthcoming on many occasions. In patients at the age of ten or thereabouts a certain number of cases of severe tumour albus show arrest of active disease, with imperfect fusion of the bones and deformity. By posterior capsulotomy through an anterior incision portion of the joint capsule at the level of the articular condyles may be divided and complete extension of the joint obtained without interference with the bones themselves. The author considers this to be the most suitable method in the treatment of severe flexional deformity of the knee, whether simple or complicated. As a preliminary measure it is wise to carry out tenotomy of the hamstrings and the *tendo Achillis*. When full extension has been restored, its maintenance can be insured by the performance of an arthrodesis.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the South Australian Branch of the British Medical Association was held at the Anatomy Lecture Theatre, University of Adelaide, on June 24, 1936, DR. D. R. W. COWAN, the President, in the chair.

Annual Report of Council.

The annual report of the Council was received and adopted. The report is as follows:

Election.

At the annual meeting held last June, officers and members of Council were elected as follows:

President: D. R. W. Cowan.
Vice-Presidents: G. H. Burnell, A. F. Stokes.
Honorary Medical Secretary: C. B. Sangster.
Honorary Treasurer: F. St. John Poole.
Ordinary Members of Council: G. Wien Smith, C. Yeatman.

At the first meeting of the Council, held on July 4, 1935, the following subcommittees were appointed:

Scientific: President, G. H. Burnell, R. A. Haste, R. D. Hornabrook, A. F. Stokes, C. Yeatman, (Honorary Medical Secretary, convener).
Contract Practice: President, R. A. Haste, A. F. Hobbs, A. F. Stokes, (Lay Secretary, convener).
Library: President, G. H. Burnell, Sir Henry Newland, L. A. Wilson, (Honorary Medical Secretary, convener).
Parliamentary Bills: President, L. C. E. Lindon, F. St. John Poole, Bronte Smeaton, (Lay Secretary, convener).
Revision of Rules: Sir Henry Newland, Bronte Smeaton, (Lay Secretary, convener).
Ethical: President, G. H. Burnell, L. C. E. Lindon, Sir Henry Newland, F. St. John Poole, C. B. Sangster, Bronte Smeaton, A. F. Stokes, L. A. Wilson, (Honorary Medical Secretary, convener).

Meetings.

The Council.—The Council has met on fourteen occasions, the attendances being:

G. H. Burnell	10	F. St. John Poole	14
D. R. W. Cowan	13	C. B. Sangster	13
R. A. Haste	12	Bronte Smeaton	11
A. F. Hobbs	10	A. F. Stokes	11
R. D. Hornabrook	12	G. Wien Smith	9
L. C. E. Lindon	5	L. A. Wilson	11
Sir Henry Newland	10	C. Yeatman	11

Scientific Subcommittee.—The Scientific Subcommittee met once, the attendance being: G. H. Burnell, D. R. W. Cowan, R. A. Haste, R. D. Hornabrook, C. B. Sangster, A. F. Stokes, C. Yeatman.

Contract Practice Subcommittee.—The Contract Practice Subcommittee met on seven occasions, the attendance being:

D. R. W. Cowan	7	A. F. Hobbs	6
R. A. Haste	7	A. F. Stokes	6

Library Subcommittee.—The Library Subcommittee met once, those present being: G. H. Burnell, D. R. W. Cowan, Sir Henry Newland, L. A. Wilson.

Ethical Subcommittee.—The Ethical Subcommittee met on five occasions, the attendance being:

G. H. Burnell	4	C. B. Sangster	3
D. R. W. Cowan	4	B. Smeaton	3
L. C. E. Lindon	–	A. F. Stokes	3
Sir Henry Newland	2	L. A. Wilson	3
F. St. John Poole	4		

Monthly General.—Nine scientific meetings were held. Interest was well maintained and attendances satisfactory. The country meeting was held in October at Victor Harbour, and a large number of members attended. A pleasant and profitable week-end was spent. The thanks of the Council are tendered to Dr. Douglas and Dr. Shipway, who helped to make this meeting such a success.

The following programme was carried out during the year:

1935.

July 25.—Paper by M. T. Cockburn: "Some Aspects of Acute Nephritis in Children". (Discussion opened by R. L. Thorold Grant.)

August 29.—Paper by D. L. Barlow: "Some Allergic Disorders". (Discussion opened by L. W. Linn.)

September 26.—Paper by Mark Mitchell: "The Relationship of Vitamins to Normal Health". (Discussion opened by F. Ray Hone.)

October 26.—Paper by A. Britten Jones: "Infection of the Hand". (Discussion opened by A. F. Hobbs.)

November 28.—Paper by W. Ray and Brian Swift: "Some Aspects of Contraception". (Discussion opened by Marie Brown.)

1936.

February 27.—Paper by P. S. Messent: "Some Aspects of Thoracic Surgery". (Discussion opened by John Hayward and Allan D. Lamphee.)

March 26.—Paper by E. F. Gartrell: "The Disturbances of the Circulatory System in Pneumonia and Other Toxic Conditions". (Discussion opened by K. S. Hetzel.)

April 30.—Paper by John Close: "Frequency of Micturition in the Female". (Discussion opened by G. H. Burnell.)

May 28.—Listerian Oration—Professor C. S. Hicks: "Non-Specific Therapy and the Vegetative Regulations of the Body".

Listerian Oration.

The oration this year was delivered by Professor C. S. Hicks on May 28, his subject being "Non-Specific Therapy and the Vegetative Regulations of the Body". There was a good attendance of members, and the Council desires to record its sincere thanks to Professor Hicks. At the close of the meeting a light supper was served in the refectory.

Membership.

The membership of the Branch is 363. The number of new members elected was 19. It is to be noted that the number of members has gradually declined since 1930, when it was 397. For the first time for five years there has been a slight increase. It is with deep regret we record the deaths of H. C. Carden, N. E. George, J. E. Good, A. A. Lendon, R. H. Pulleine and C. E. Dolling.

Representation on Boards.

Dental Board.—P. S. Messent.
Nurses' Board.—S. R. Burston.
Medical Board.—H. H. E. Russell.
Metropolitan Infectious Diseases Hospital Board.—H. H. E. Russell, P. T. S. Cherry.

Work of Sections.

Eye, Ear, Nose and Throat Section.—Eight meetings were held during the year, the attendances being satisfactory.

Clinical Medicine.—Four meetings were held, the average attendance being 20. The membership is 78.

Surgery.—Four meetings were held during the year, and the average attendance has increased from 20 to 25. The membership has increased to 42 members.

History of Medicine.—Four meetings were held, two taking the form of illustrated lectures. The attendances at the meetings have been disappointing, the high standard of the papers and addresses meriting stronger support from members.

Lodge Practice.

There has been no alteration in the conditions of lodge practice, but the Council has under consideration the desirability of reverting to the original terms agreed upon for family attendance and the age limit for juveniles. During the depression, in order to afford relief to family lodge members, the rate was reduced from 10s. 6d. to 9s. per quarter and the age at which juveniles could remain on the juvenile list raised from 16 to 18 years. The arrangement was for one year only, but it has remained in force for four and a half years, and it is felt that the time is opportune to return to the terms of the original agreement.

Despite every attempt to secure uniform rates and conditions for the treatment of lodge patients by eye, ear, nose and throat specialists, success has not been achieved. Whilst the principle of uniform rates and conditions has been accepted by the lodge representatives, no agreement could be reached in regard to those rates and the Council has reluctantly felt it would be useless for the present to proceed any further in the matter. It is still felt, however, that uniformity of rates and conditions is desirable and that it may be possible to introduce them at some future time.

Adelaide Permanent Post-Graduate Committee.

The series of twelve lectures delivered from May to August, 1935, was well attended. The usual post-graduate course was held during May of this year. Dr. Allan S. Walker, of Sydney, delivered the "E. C. Stirling Lectures", and a considerable number of members availed themselves of the opportunities provided by the Committee. This Committee, which is at present a subcommittee of the British Medical Association, has done excellent work, and it is thought that the time has come when its constitution should be more clearly defined. The matter is receiving consideration, but no finality has yet been reached.

Control of Tuberculosis in South Australia.

The position in regard to the control of tuberculosis still leaves much to be desired. The clinic at the Adelaide Hospital is now functioning, but no satisfactory results are likely to accrue until closer cooperation between the Health and Hospital Departments is arranged and someone is made responsible for the whole tuberculosis organization. This matter is still receiving the attention of Council.

Revision of Rules.

It is hoped that this matter will soon be finalized. Correspondence has passed between the Council and the Parent Association, which made several suggestions concerning the rules. These have received the attention of the solicitors to the Branch, and it is expected that shortly the rules will be approved by the Parent Association. As soon as this is done it is the intention of the Council to incorporate the Branch under *The Associations Incorporation Act*, as advised by the Branch solicitors.

Annual Meeting of the Parent Association.

An event of outstanding importance in the history of the British Medical Association in Australia was the annual meeting of the Parent Association held at Melbourne in September, 1935.

The Victorian Branch is to be congratulated on the splendid success which attended its efforts. A strong representation of South Australian members attended the meeting, and subsequently this Branch was privileged to entertain Lord Horder, Professor Hercus, Mr. F. F. Muecke and other distinguished visitors to Adelaide.

The Council suggested that, as the expenses of the Victorian Branch would be very heavy in connexion with the annual meeting, it would be a graceful act if members contributed amounts to assist in this direction. A satisfactory response was given and the Victorian Branch has expressed its sincere appreciation of the consideration shown by members of this Branch.

Australasian Medical Congress (British Medical Association), Fifth Session, 1937.

The fifth session of Congress will be held in Adelaide from August 23 to 28, 1937. On the recommendation of this Branch, the Federal Council has appointed Sir Henry Newland to the position of President. The whole of the members of the Branch have been constituted the General Committee of Congress. The Executive Committee has been formed and has already commenced active organization. Endeavours are being made to secure a strong delegation from overseas, and it is confidently expected that the Congress will maintain the high standard of previous ones. Success will depend largely on the support given and the interest shown by members in the work of the Congress. To stimulate the interest of members and let them know what is being done, it is proposed about August next, when the organization is more advanced, to hold another meeting of the General Committee of Congress.

Emergency Treatment to Persons Injured through Road Accidents.

Representations have been made by the Council to the "Traffic Committee" that in any new regulations adopted some provision should be made for payment to medical practitioners for emergency treatment in road accidents. Notification has been given in the Press that in the Bill being drafted this has been done. It has not been possible to obtain a draft of the Bill, but members will be glad to learn that the thankless burden borne by the medical profession for many years in rendering emergency treatment for those injured on the roads will in all probability be removed.

Ministry of Health.

Following a report presented to the Government containing suggestions for a reorganization of the Health Department, a deputation waited on the Chief Secretary to urge the recommendations submitted. A reply was received in August stating that no change was contemplated or desired for the present. Later an attempt was made to interview the Premier to insure that the Government was fully advised regarding the suggestions made. The Premier, however, replied that the matter was being considered by the Chief Secretary, who was the Minister of Health, and he was unable to receive a deputation from the Council. With a view to placing on record that an earnest attempt had been made by this Association to put health matters on a better footing, it was decided to publish the report in *The Advertiser*. This was done. It is a matter for regret that in this, as in many similar instances during the past few years, the Government has elected to ignore recommendations and reports of this Association.

Rations Supplied by the Government to Children of Unemployed.

In June last a request was received from the Government asking the Association to assist in determining what is an adequate ration allowance for children up to the age of fourteen years. A subcommittee was appointed by the Council to investigate and report on the matter, and as a result of the inquiry a report was forwarded to the Government in July last. The recommendations contained in the report were adopted by the Government.

Federal Council.

Two meetings of the Federal Council were held during the year, the first being held at Melbourne in September, 1935, and the second in Sydney in March, 1936. Our representatives, Sir Henry Newland and Dr. Bronte Smeaton, attended both meetings.

Contributions in Health Notes.

In an endeavour to foster close and amicable relationship between official health authorities and private practitioners, the Council was asked by the Central Board of Health to contribute articles in the pamphlet, *Public*

Health Notes, a quarterly publication. This was agreed to, and articles have since appeared in the pamphlet contributed by the Council of the South Australian Branch of the British Medical Association.

Medical Officers to the Destitute.

It was the intention of the Council to again consider the remuneration paid to medical officers to the destitute, and members were asked to supply information to support a further approach to the Government. Owing, however, to the lack of assistance received from medical officers, the Council was unable to take any further action in the matter.

Medico-Legal Society.

During the year a Medico-Legal Society has been formed. Fifteen members notified their intention of joining the society. The inaugural meeting was held on May 5 last.

Home for the Branch.

The Council has appreciated the necessity of a permanent home for the Branch. In other States the Association has its own building, but the South Australian Branch is dependent on the University for accommodation to hold its meetings. The British Medical Hall Company has had under consideration for some time a scheme for the erection of a building on the North Terrace property of the Company and has in hand plans which will provide accommodation for the Branch. At a later date this Association will be approached in order to ascertain if it approves and will support the scheme put forward by the Hall Company.

Honour to Members.

The Council tenders its congratulations to Dr. Helen Mayo, O.B.E., Dr. A. M. Cudmore, C.M.G., and Sir Stanton Hicks on the honours conferred upon them.

Inspector-General of Hospitals.

During the year one of our members, Dr. L. W. Jeffries, was appointed to the responsible position of Inspector-General of Hospitals. This Branch offers its sincere congratulations to Dr. Jeffries and feels confident that the happiest relations will be maintained between the Association and the Inspector-General's Department.

Death of King George V.

The Council forwarded through the Government a message of sympathy to His Majesty the King, Queen Mary, and members of the Royal Family on the death of His Late Majesty King George V. A letter has been received from His Excellency the Governor (Sir Winston Dugan) on behalf of His Majesty, Queen Mary, and other members of the Royal Family, conveying to the Council an expression of deep appreciation of the loyal message of sympathy.

General Remarks.

Though we have not attained all the objectives aimed at during the year, some progress has been made. It is perhaps well that reforms should come about gradually, so that errors, as far as possible, may be avoided. There is plenty of work yet to be done, and it is expected that members will accord to the incoming President and Council their whole-hearted support, so that this Association may continue to maintain the high position in the counsels of the community to which its important and special knowledge entitles it.

In conclusion, I wish to thank the Council and members for their loyal support during the year, and the Lay Secretary for his untiring efforts in the interests of the Association.

(*Signed*) D. R. W. COWAN,
President.

Annual Statements.

The financial statements were presented by the Honorary Treasurer, Dr. F. St. John Poole, and adopted. The financial statements are published herewith.

Income and Expenditure for year ended December 31, 1935.

	£ s d.	£ s. d.
To British Medical Association, London	459 6 0	
„ THE MEDICAL JOURNAL OF AUSTRALIA	334 0 0	
		793 6 0
„ Library Subscriptions		60 15 0
„ Rent	55 19 0	
„ Telephone	40 14 10	
„ Stationery and Printing	55 0 10	
„ Postages and Telegrams	31 13 3	
„ Exchange	3 2 5	
„ General Expenses	45 10 1	
		232 0 5
„ Listerian Oration Expenses		9 16 6
„ Lister Medal		0 10 0
„ Salary		350 0 0
„ Federal Council		31 14 0
„ Depreciation		9 0 0
„ Balance		16 0 8
		£1,503 2 7

	£ s d.	£ s. d.
By City Subscriptions	867 11 0	
„ Country Subscriptions	397 16 6	
„ Accrued Subscriptions	219 5 0	
		1,484 12 6
„ Interest		5 10 11
„ Accrued Interest		2 19 1
„ Medical Certificate Books		0 4 2
„ Lister Fund		9 15 11
		£1,503 2 7

General Fund Account.

	£ s. d.
To Balance	2,858 2 4
	£2,858 2 4

	£ s. d.
By Balance brought down, December 31, 1934	2,842 1 8
„ Income and Expenditure Account	16 0 8
	£2,858 2 4

Library Fund Account, December 31, 1935.

	£ s. d.	£ s. d.		£ s. d.
To Adelaide University	50 0 0		By Balance brought down, December 31, 1934	510 5 5
„ Adelaide University, Special Donation	15 0 0		„ Interest from Savings Bank	2 6 7
		65 0 0	„ Transfer from Income and Expenditure Account	60 15 0
„ Depreciation		9 3 11		
„ Balance		499 3 1		
		£573 7 0		£573 7 0

Balance Sheet as at December 31, 1935.

Liabilities.	£ s. d.	£ s. d.	Assets.	£ s. d.	£ s. d.
To Subscriptions Paid in Advance		20 11 0	By Plant and Fittings	90 0 0	
„ British Medical Association, London—			*Less* Depreciation	9 0 0	81 0 0
Paid Subscriptions	168 18 9		„ British Medical Hall Company, Limited		2,060 0 0
Unpaid Subscriptions	67 11 6		„ Library Fund—		
		236 10 3	Delineascope	36 19 11	
„ The Medical Journal of Australia—			Projector	52 0 0	
Paid Subscriptions	126 10 0			88 19 11	
Unpaid Subscriptions	53 0 0		*Less* Depreciation	9 3 11	
		179 10 0			79 16 0
„ Library Fund		499 3 1	„ Savings Bank Library Fund ..		73 6 1
„ Sundry Creditors		1 15 0	„ Lister Medals and Dies		18 3 4
„ General Fund		2,858 2 4	„ British Medical Hall Company, Limited—Loan Account ..		450 0 0
			„ Subscriptions owing		322 0 7
			„ Stocks—		
			Hospital Forms	0 15 7	
			Medical Certificate Books ..	3 9 0	
			Stationery	10 0 0	
					14 4 7
			„ British Medical Hall Company, Limited	151 6 4	
			„ Sundry Debtors	7 13 1	
					158 19 5
			„ Cash—		
			Savings Bank of South Australia	298 9 4	
			Commonwealth Savings Bank	151 11 9	
			National Bank of Australasia Limited	25 7 9	
			In Hand	62 12 10	
					538 1 8
		£3,795 11 8			£3,795 11 8

I certify that I have examined the books and vouchers of the British Medical Association as produced to me for the year ended December 31, 1935, and that in my opinion the above Balance Sheet correctly sets forth the financial position of the Association as at the above date and as shown by the books.

C. W. L. MUECKE,
Chartered Accountant (Aust.),
Auditor.

Adelaide,
May 14, 1936.

F. ST. JOHN POOLE, Honorary Treasurer.
WALTER C. DOBBIE, Secretary.

Introduction of President.

Dr. D. R. W. Cowan then introduced the President for the ensuing year, Dr. A. F. Stokes, and wished him a successful term of office.

Dr. A. F. Stokes thanked the members for having elected him to the presidential chair.

Election of Office-Bearers.

Office-bearers for the ensuing twelve months were elected as follows:

Vice-President: Dr. R. E. Magarey.
Honorary Medical Secretary: Dr. C. B. Sangster.
Honorary Treasurer: Dr. F. St. John Poole.
Members of Council: Dr. P. T. S. Cherry, Dr. C. F. Pitcher, Dr. O. M. Moulden, Dr. E. F. Gartrell.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 25, 1936, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. E. H. M. STEPHEN, the President, in the chair.

Bronchiectasis.

DR. M. J. PLOMLEY read a paper entitled: "Bronchiectasis" (see page 116).

DR. A. B. K. WATKINS read a paper entitled: "Oto-Rhino-Laryngological Considerations in Bronchiectasis" (see page 118).

DR. H. L. DALY remarked that Dr. Watkins had said that there was a wave of enthusiasm in America for bronchoscopic methods for investigation and treatment of patients suffering from bronchiectasis. Dr. Daly had visited Chevalier Jackson's clinic in Philadelphia, where he saw bronchoscopic aspiration performed on children with diseases of the lungs. He had been amazed at the ease with which these children were examined. They lay quietly and could be thoroughly examined, although no preliminary sedative was given. The oro-pharynx was sprayed with 10% cocaine, a hypodermic injection of one one-hundredth of a grain of atropine was given to obtain a dry field and then the patient was brought in for operation. Jackson used instruments that were not metal-plated because, he said, from plated instruments the light shone back into the eyes of the operator and might temporarily blind him. The covers on tables, gowns and caps used at operation were green, because it was complementary to the red colour of the bronchial tree; it followed that when the operator looked down the laryngoscope he could see clearly lesions which otherwise might be missed.

Dr. Daly said that he had used lipiodol and he was still uncertain whether it was better to use descending or ascending lipiodol. He usually employed the descending form. He had originally introduced the lipiodol by puncturing the crico-thyreoid membrane, but on one occasion he had infected the tissues of the neck while he was withdrawing the needle. Since then he had cocainized the larynx with a 10% solution of cocaine; he then passed a catheter with the aid of a laryngoscope and thus injected the lipiodol into the lungs. The patient should be turned on either side and each lung field filled separately before taking the X ray picture, so that all trauma was avoided.

Dr. Daly had seen lobectomy performed at Wisconsin University Hospital, Madison, with "Cyclopropane" as the anæsthetic. "Cyclopropane" had the advantage that 80% of oxygen could be given with 20% of this gas. With this large amount of oxygen the patient could be kept a good colour and under control. It was the ideal anæsthetic for operations on the lungs. Dr. Daly had been surprised at the extent to which the ribs could be separated and the good view of the lung which could thus be obtained. Dr. Daly had also seen lobectomy performed by Tudor Edwards and Nelson at Brompton Hospital, London. The ease with which these men worked in the thoracic cavity itself was remarkable. At this time they had used gas and oxygen anæsthesia, but Dr. Daly thought that they now used "Cyclopropane". McGill, in a recent journal, said that between 1922 and 1930 he had given only six anæsthetics for lobectomy, but from 1930 to the present time he had given 126. The thoracic surgeon was coming into his own. In conclusion Dr. Daly said that early treatment, often surgical, should be insisted on in order to get the best results in bronchiectasis and allied diseases of the lung.

DR. E. P. BLASHKI said that he was grateful for the trouble to which Dr. Watkins and Dr. Plomley had gone in the preparation of their papers. He did not intend to discuss the causation of bronchiectasis. He wished to point out the frequency of sinusitis in children and the possibility of recognizing it at an earlier age than usual. Bronchiectasis frequently followed whooping cough, measles *et cetera*; sinusitis was a frequent accompaniment of these diseases as well. Scarlet fever rarely occurred without sinusitis. Many sufferers from the exanthemata were allowed to go out of the doctor's care with a sinusitis. Dr. Blashki said that it had been his lot to see many of these patients in early childhood with well-established sinusitis and profuse sputum. The conditions must be looked for at an early stage and attacked without delay.

Dr. Blashki said that work done in Melbourne showed that many patients with lobar pneumonia had also antral disease. Lavage of the antrum was beneficial in the cure of the pneumonia. Bronchiectasis was progressive and therefore caution must be used in selecting cases for gross operative treatment. It should never be undertaken without radiological examination with lipiodol. Dr. Blashki said that it took a long time to establish a cure; it was a question sometimes of years and not months. Time must be taken to treat both the sinus disease and the lung. Postural drainage had to be constant and systematic, and bronchial drainage could be undertaken to supplement it. But so long as the bronchi could be drained well posturally, postural drainage should be persisted in. It was impossible to maintain adequate drainage with the bronchoscope alone. Some of Dr. Blashki's patients at the Royal North Shore Hospital did well with occasional drainage and found it so beneficial that they came back for a repetition of the procedure. A mixture of "Ti-trol" and oil was useful when there was fœtid odour. A solution of from four to five cubic centimetres of oil with 1% to 2% "Ti-trol" kept the patient free from odour.

Dr. Blashki thought that the success of vaccines depended on the material's being obtained directly from the lung. He referred to one case in which rapid cure had followed the preparation of a vaccine of *Bacillus pyocyaneus* isolated from the lung.

Dr. Daly had expressed doubt as to whether the lipiodol used should be the ascending or descending variety. Dr. Blashki said that the descending lipiodol was the one to use. With ascending lipiodol no shadow was obtained, because there was not sufficient iodine content.

Bronchiectasis was best treated by constant and complete cooperation when the same people worked together as a team. Intermittent work did not give continuity of interest nor benefit to the patient.

DR. COTTER HARVEY said that he had been very interested in both papers; each had the merit of bristling with points for discussion.

Dr. Plomley had stated that fibrosis was the basic factor in bronchiectasis. The essential cause of bronchiectasis was, however, not so simple, nor was it yet fully understood. Broadly speaking, there were three theories as to the cause of bronchiectasis. First, the traction theory, with fibrosis as the essential; this was not satisfactory, as it presumed that the fibrous tissue was anchored both at mediastinal and pleural surfaces, and as it contracted it caused dilatation of the bronchi. But in many cases the pleura was free, as was proven by the successful induction of artificial pneumothorax. The second, or obstruction, theory was based on the belief that internal expansion of the bronchus occurred with coughing. To this the objection was raised that a cough was not an expanding but a compressing force on the pulmonary tissues. The third theory was that of "compensation"; this postulated the dilatation of the bronchi as a compensatory mechanism whereby a vacuum was prevented. This was worthy of careful consideration and was probably the most important cause in children, following bronchopneumonia. Here there occurred patchy collapse of small areas of the lung. If, as might happen, collapse persisted, it was necessary for other areas of the lung to take up the space. Thus the soft-walled bronchi in children underwent dilatation.

Sinus infection, as Dr. Watkins had said, introduced the "wet" element. It might be helpful to distinguish between

cases of bronchiectasis in which sinusitis was present and those in which it was not. Dr. Harvey had been surprised at the high percentage of cases in which sinus infection had been present in Dr. Watkins's series; it had seemed to him an extraordinarily high figure. He had therefore undertaken an analysis of forty proven cases of bronchiectasis from the clinic at the Royal North Shore Hospital and had found that sinus infection was present in twenty, or 50%. In patients under twenty years of age the percentage was 63, a figure not far below that of Dr. Watkins.

Dr. Harvey wished to emphasize that true lobar pneumonia did not lead to bronchiectasis. The damaging pneumonias of childhood were of the patchy catarrhal type, such as those complicating the exanthemata. Diagnosis could really be very difficult in bronchiectasis. There were three types of patients: (i) those with obvious signs and symptoms of bronchiectasis; (ii) those who had suggestive symptoms, but who were shown by lipiodol to have an intact bronchial tree; (iii) those who had neither signs nor symptoms, except when a chance hæmoptysis led to the discovery by lipiodol bronchogram of dilated bronchi.

In regard to the relationship between cerebral abscess and bronchiectasis, Dr. Harvey adhered still to the metastatic theory. He would cite three examples in support of this theory: (i) In bronchial carcinoma cerebral metastases were not at all uncommon; they reached the brain through the blood stream as emboli, and the same path was followed by a septic embolus. (ii) He had once had under his care a patient with a chronic empyema. This patient developed hemianopia and severe headache and died shortly after from a suppurative meningitis. At *post mortem* examination an occipital lobe abscess was revealed. There was no doubt as to the mode of origin in this case. (iii) There had recently died in the Royal Prince Alfred Hospital one of his patients with bronchiectasis complicated by a frontal lobe abscess. The onset of the latter was marked by a typical apoplectic seizure, the patient being admitted to hospital unconscious, with a hemiplegia. This, as Dr. Watkins had stated, was very suggestive of an infective infarct.

Dr. Harvey thought the percentage of cases quoted by Dr. Watkins as being diagnosable without lipiodol was too high.

Dr. Harvey said there was still need for a great deal of clinical research on this subject. He would have liked, for instance, to have heard from Dr. Plomley of some follow-up of those cases he had seen among his outpatients; this would have been very informative. There were several questions to which he would like to find an answer. How long did bronchiectasis take to appear and was it necessarily progressive? Or could it heal spontaneously? In the forty cases analysed the condition had persisted on the average for a period of ten years. Again, why in some patients did it occur so easily while in others with apparently identical insult to their pulmonary tissues no apparent lesion, or at most chronic bronchitis, resulted. There might be some congenital factor that determined this. Many children in this country had sinusitis, but comparatively few had bronchiectasis. Why was this?

In the last two patients that Dr. Harvey had seen who had been submitted to lobectomy the end result had been unsatisfactory. One had developed bronchiectasis in the opposite lung and the other a similar condition in the lower part of the remaining upper lobe on the same side. Surely some patients were particularly prone to bronchiectasis while others would never develop it.

Dr. Harvey endorsed what Dr. Blashki had said concerning the need for earlier treatment and for prophylaxis in measles and the other causes of bronchopneumonia. Bearing on what he had stated earlier, he thought that an important preventive measure in children might be insistence on deep breathing exercises during their convalescence and for some months afterwards.

DR. GARNET HALLORAN thanked the speakers for the manner in which they had presented their papers. He spoke of the relationship between sinusitis and bronchiectasis. The patient with the slushy cough might have a comfortable life after thorough eradication of the sinus disease. It was interesting to see that six months after operation some patients were no longer spitting up pus, but a mucoid sputum. Dr. Halloran often sent such patients to the New England ranges.

In early bronchiectasis in some children the cough and sputum appeared to cease completely after sinus operation. When chronic sinusitis was known to be a factor, operation could be undertaken earlier. It was interesting to see the increased desire of physicians for pneumography; they wished to know which lobe, whether only one lobe or more than one was involved. This had been increasingly evident during the past year, and Dr. Halloran hoped that it would lead more cases to the lobectomist.

In 1920 Professor A. E. Mills had taught and written on chronic bronchitis due to empyema of the antra. His teaching inspired a system by which children were sent to the X ray department to have skiagrams taken of their skull sinuses and chests. Such a system, with the later addition of lipiodol, became firmly established.

Dr. Halloran thought that when the characteristic slushy cough was heard the medical attendant should think in terms not only of the stethoscope, but also of the sinuses, especially the antra.

DR. C. G. MCDONALD said that he had listened to the two papers with pleasure. He felt like coming to Dr. Plomley's defence on the question of the part played by infections of the lung in the production of bronchiectasis. The great majority of serious cases of bronchiectasis originated in pulmonary infections, which were not always obvious and were often not recognized, but of which there was frequently radiological evidence after the patient's recovery. Dr. McDonald said that in his experience the worst cases were those following pneumonia and other pulmonary infections.

The ear, nose and throat specialists thought that sinusitis and bronchiectasis were so closely connected that one rarely occurred without the other, and some of them believed that bronchiectasis resulted from sinusitis, which was the primary causal condition. Dr. McDonald did not agree with these views. He was of opinion that when sinusitis and bronchiectasis existed together, they represented the aftermath of a primary infection of the whole upper respiratory tract. The bronchial and sinus infections were the residuum of this generalized infection. The improvement manifested by the bronchiectatic patient after antrostomy was due to removal of a coexisting infection and not to removal of the alleged cause. Theoretically an infection might spread from the accessory sinuses of the nose down the bronchial tree and lead to bronchiectatic dilatation. Influenza was a disease in which an infection of the naso-pharynx and trachea spread directly into the bronchi and the pulmonary structure. But such a spread occurred rapidly and at the height of the infection. Dr. McDonald thought that the rhinologist's tendency to regard sinusitis and bronchiectasis as cause and effect was an example of the danger of specialization. The rhinologist had a special knowledge of sinusitis, but no special knowledge of bronchiectasis. The main objection to this view was that in so many cases of chronic tonsillar and naso-pharyngeal infection there was no sign of bronchitis and certainly no evidence of bronchiectasis. What was bronchiectasis? Dr. McDonald had seen the diagnosis made on the slenderest evidence, frequently on radiological evidence alone. There might be a report by the radiologist of the existence of bronchiectatic lesions of which there was no clinical evidence. The radiographic diagnosis of bronchiectasis was often made on the appearance of cylindrical markings, which were possibly part of the normal pulmonary reticulum or of the exaggerated peribronchial markings which occurred as a result of respiratory infection in earlier life.

Dr. McDonald had been interested in Dr. Watkins's paper, which was very thoughtful, as were all his papers. Dr. Harvey had taken Dr. Watkins to task for saying that cerebral abscess complicating bronchiectasis was not due to metastatic infection from the lungs, but possibly to direct spread from the sinus infection. Dr. McDonald

would also take him to task, but not on the same grounds as Dr. Harvey. What Dr. Watkins had said was true. It was difficult to understand the occurrence of cerebral abscess as a result of septic embolism from the lung when so little metastatic involvement occurred in other parts of the body. In subacute bacterial endocarditis emboli might occur in the spleen and liver and kidneys, and anywhere at all, including the brain, but this generalized metastasis was rarely seen in bronchiectasis. At the same time it was difficult to understand the suggested causal relationship between sinus suppuration and cerebral abscess in cases of bronchiectasis, since the frequency of cerebral abscess in ordinary cases of sinus suppuration not associated with bronchiectasis was low. There was possibly another explanation. Perhaps in bronchiectasis the infection travelled from the hilus of the lung into the lymphatics of the neck and so to the cerebral vessels, just as malignant disease of the mediastinum frequently spread into the glands at the root of the neck. This could conceivably occur in bronchiectasis without frank infection of the cervical glands manifesting itself. The problem of cerebral infection, however, was not yet solved, and Dr. Watkins had done good service by calling attention to the difficulties.

DR. LAURENCE HUGHES said that he wished to take up the gauntlet with Dr. Harvey concerning the follow-up of cases at the Royal Alexandra Hospital for Children. Dr. Hughes had been interested in the subject of bronchiectasis in children for several years. Though the treatment of bronchiectasis had advanced rapidly, Dr. Hughes wondered whether everything possible was being done in the main line of treatment, namely, prophylaxis. This might seem a platitude. For the past four years Dr. Hughes had made a point of examining clinically and radiologically all children with a chronic cough who had a history of whooping cough and measles as one group and of pneumonia as another. As a result of clinical and radiological examination he had found that a fair percentage of these patients had bronchiectasis. Was the profession as a whole doing all that it might to prevent the incidence of this disease? Dr. Hughes stressed that in all cases of measles and whooping cough and pneumonia in children there should be as efficient a supervision of convalescence as possible. The sinuses should be examined radiologically as a routine measure and the patients, if necessary, should be referred to the ear, nose and throat department. A fair percentage of patients had sinuses that required treatment. But quite a number had no sinusitis. Dr. Hughes thought that sinusitis was important, but in a fair percentage of patients there was no evidence of it, even though the patients certainly had bronchiectasis.

Dr. Hughes, in conclusion, said that from experience with the patients he had followed up at the Royal Alexandra Hospital for Children, he was of opinion that the prognosis in cases of definitely established bronchiectasis was not good. But minor degrees of the condition could be ultimately cured.

DR. K. E. SHELLSHEAR said that he had been interested for years in sinus and chest affections in children. In his experience the percentage of infective sinuses was at least 60%; and this was a low estimate in the cases that he had seen.

DR. D. G. CARRUTHERS said that Dr. Harvey had touched on a point that had exercised his mind. Why did so many patients with bronchiectasis also have sinusitis; yet why were there so many patients with sinusitis but without bronchiectasis?

For some time now he had performed antral lavage on the majority of his tonsillectomy patients, both adults and children, and found about 20% to have given positive pus washings, yet less than 1% of these patients had bronchiectasis. Probably, then, there was some third factor to explain the onset and persistence of both the bronchiectasis and the sinusitis in these cases. The amount of pus in either the antra or the lungs could be diminished by draining it away, but the condition in either site was very resistant and hard to cure when sinusitis and bronchiectasis were coexistent. In such cases there might possibly be a biochemical factor, maintaining the infection in both sites.

DR. E. H. M. STEPHEN described briefly an apparatus he had seen at the Children's Hospital, Melbourne, for postural drainage of patients with bronchiectasis. The patients eventually spent the entire night asleep on it. He also described a special stretcher in use at the Royal North Shore Hospital of Sydney, which he had seen at the recent clinical meeting held there. This stretcher successfully tilted the patient up without any fear of his slipping.

Dr. Harvey said that this apparatus, spoken of by Dr. Stephen, had been copied from the Austin Hospital, Melbourne. He then proceeded to show a series of films.

Dr. Plomley, in reply, said that he had been told that he had not sufficiently emphasized the difficulty of diagnosis of bronchiectasis. It had been said that in some cases diagnosis was easy, but in many it was difficult. Diagnosis in the dry case needed much confirmation, and if the practitioner was in doubt he had to get the help of an X ray picture. There were certain cases which might be mistaken for bronchiectasis, for instance fœtid bronchitis. This condition was acute and went on to a fatal result, but Dr. Plomley did not think that bronchiectasis was present in spite of the similarity of symptoms. Dr. Plomley referred to the question of cure in acute cases in which the cause was removable; in such instances cure was possible. He thought that cure was also possible when the secretions could be dried up; even if there was fibrosis the child might be cured—at any rate as regards symptoms. At the Royal Alexandra Hospital for Children, children were not treated after the age of twelve years and it was impossible to follow them up. He knew of several cases in which the acute bronchiectasis dried up and the patient got well.

As to the question of sinus infection, Dr. Plomley wondered how old was the youngest child in whom sinus infection occurred and how old the youngest in whom bronchiectasis occurred. He was sure that in many children with bronchiectasis sinusitis was not the cause.

Dr. Watkins thanked the speakers for treating him in such a tender manner. Dr. Daly had said that Chevalier Jackson acknowledged that he did not have his instruments plated. Dr. Watkins understood that this was because the plating peeled off bronchoscopes. The edges left by the plating might be sharp; therefore the instruments were better unplated.

In regard to Dr. Daly's advocacy of catheterization, Dr. Watkins said that his method was to anæsthetize the larynx and lower part of the nose and to pass a Jacques catheter through the nose; then by indirect laryngoscopy it was possible to see what he was doing and, by tilting, the head of the catheter could be guided into the larynx. After insertion the catheter was fixed to the side of the cheek with strapping, and a lipiodol injection was given immediately before the X ray picture was taken.

Dr. Plomley had asked what age was the youngest child with bronchiectasis. Dr. Watkins's youngest patient had been two and a half years. When the child had been turned upside-down one and a half pints of pus were evacuated. This had been a terrific demonstration.

Concerning cerebral abscess and bronchiectasis, Dr. Watkins admitted that he did not know the relationship, though he believed that the majority of abscesses were caused by sinusitis. He had been interested in Dr. Harvey's cases, especially the two of metastases resulting in brain abscess from other causes. Carcinoma of the lung with metastases had a tendency to send out blunderbuss secondary deposits and it would be surprising if some of these did not find their way to the brain.

Dr. McDonald had overstated the case when he had said that the ear, nose and throat men said that sinusitis and bronchiectasis did not occur apart. Sinusitis was common, while bronchiectasis was rare. At the Newcastle Hospital the conditions had occurred together in only fifty cases in the series referred to. But during the same period there

were thousands of cases of sinusitis without bronchiectasis. This was what Dr. McDonald meant to say, but no doubt he had overstated for the sake of emphasis.

Dr. Stephen said that he was glad to be able to thank the speakers and also those who had taken part in the discussion. The two papers were admirable contributions to the subject.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Jobson, Philip Latham, M.B., B.S., 1935 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Practice.

THE ADELAIDE RADIOTHERAPY CLINIC.

THE following circular has been issued from the Radiotherapy Clinic at the Adelaide Hospital.

CANCER OF THE TONGUE.

At a meeting of the Adelaide Hospital Radiotherapy Clinic held on April 21, 1936, the subject of discussion was cancer of the tongue.

Précis of Literature.

Investigation of a subject such as this naturally falls into two main stages: (i) consideration of the primary lesion, and (ii) consideration of the glandular deposits. For convenience these two stages are, as far as possible, kept separate.

The Primary Tumour.

Members of the Paris school prefer radium treatment by the insertion of needles into the tongue when the lesion is situated in the anterior two-thirds. For tumours of the floor of the mouth they recommend radium application by means of a moulded apparatus, and for tumours of the posterior third of the tongue insertion of needles in combination with external radiation by means of a radium "bomb" or deep X rays. The three-year cure rate quoted by Roux-Berger improved from 11·3% in 1920 to 32·7% in 1924 and 38·9% in 1925; the improved results were ascribed to improvement in technique. The cure rate for 287 patients treated between 1920 and 1925 was 26·8%. The important rôle of glandular deposits in causing death was shown by the fact that actually 46·3% of the patients showed healing of the primary tumour, the deaths from deposits reducing the survival figure to 26·8%.

Stanford Cade, in the 1934 annual report of the Westminster Hospital, also pointed out the necessity for needling tumours in the anterior two-thirds of the tongue; he uses a moulded apparatus for treating lesions of the floor of the mouth and strongly favours the use of a radium "bomb" for tumours of the posterior part of the tongue and the pharynx. In his Hunterian lecture in 1933 (*The Lancet*, Volume II, 1933, page 4) Cade remarks:

> Some of the failures in radium therapy need not be blamed on the agent itself, but on the operator, who fails to take advantage of up-to-date methods, who uses radium occasionally, who has only a vague idea of dosage, intensity and biological effects, and to whom the natural history of the disease is a closed book.

In discussing treatment he says that in early lesions of the anterior portion of the tongue it matters little whether radium or surgery is used—in fact, competent surgery is infinitely to be preferred to incompetent radiation treatment. He quotes Berven's results after radium treatment in operable lesions (59·1% cures after three years) and compares them with the results after surgery (20% to 25% for a similar period, quoted by Lane-Claypon in a Ministry of Health report).

Norman Patterson (*The Lancet*, Volume II, 1934, page 633) recorded in 1934 his method of treating tumours of the mouth with diathermy. He states that most of his patients were referred in a fairly advanced stage and were regarded by surgical colleagues as being "inoperable". Eight out of 31 consecutive patients, or 26%, were alive five years after treatment.

It was interesting to note the paucity of statistical tables covering the results of surgical treatment of carcinoma of the tongue.

Glandular Deposits.

The French practice in the treatment of cervical lymph glands is summarized in articles of Roux-Berger and Regaud, both of whom recommend a radical dissection, based on Crile's operation, for every patient in whom removal of these glands is practicable. Since replacing general by local anæsthesia for this operation, Roux-Berger has not had a death from bronchopneumonic complications. He finds that the extent of glandular invasion is always greater than clinical examination would have led him to expect, and he is becoming more and more radical in his treatment when glandular enlargement is little or absent, and less and less radical when massive deposits have occurred. He finds that in the latter case external radiation treatment alone gives greater palliation than any combination of treatments, including an attempt at surgical ablation. Regaud points out that at least 60% of patients suffering from cancer of the lip do not have deposits in the glands and that a routine excision of glands in this disease is therefore unnecessary. In cancer of the tongue, on the other hand, only 25% to 30% of sufferers are free from such deposits and a routine excision becomes necessary. In a later article Regaud discusses the causes of enlargement of palpable cervical lymph glands. He points out that in only 60% of cases of cancer of the lip do the enlarged glands show microscopic evidence of deposits, while this number rises to 80% when cancer of the tongue is considered.

Other authorities, including Quick (*American Journal of Roentgenology*, Volume XXXIII, page 667) favour interstitial irradiation of cervical lymph glands, especially when the operability is in doubt. This treatment, although combined with external irradiation, is thought to be more effective than the latter alone.

The most widely held opinion is that a radical Crile's dissection is the main treatment of gland areas draining a carcinoma of the tongue.

Experiences at the Adelaide Hospital.

Fifty-five patients suffering from cancer of the tongue have been treated in the radiotherapy clinic at the Adelaide Hospital during the past six years. Of the twenty-nine patients available for investigation three years after treatment it is found that seven, or 24·1%, are alive. Survivals were practically confined to patients in whom the lesion was in an early stage. Many of the lesions were in an advanced stage, three of the earlier patients being referred with inoperable glandular deposits after amputation of the tongue. For over six years no patient in the Adelaide Hospital has been treated for carcinoma of the tongue without the use of radium or deep X rays, although in several cases use has been made of diathermy-coagulation in addition. In this connexion it must be remembered that the method of treatment is invariably decided by the consultative clinic after full discussion and that each unit of the surgical staff has a representative on the clinic.

Deep X ray treatment has been tried with certain advanced lesions, particularly those of the most posterior part of the tongue. In several patients an excellent palliative result has been obtained, but up to the present we have been unable to produce a cure by this method.

The treatment of glandular deposits has been shown to be a different matter, for none of our patients with proved deposits in the cervical glands has lived as long as two

years after operation, and no cures by radiological means alone have been recorded. Analysis of the 23 operations for removal of cervical glands showed that only five had approached a Crile's dissection, the remainder being less radical.

Syphilis is probably an important factor in the genesis of cancer of the tongue, for of the 38 patients in whom the Wassermann test was made, eight, or 21·1%, gave a positive reaction. This may be compared with the 3% or 4% of syphilitics among patients with cancer of the lip. Treatment of this group of eight patients has on the whole been followed by an unusual degree of pain. In one of them the tumour was radio-resistant. Extensive glossitis in others has made it difficult to be certain that the tumour has been eradicated.

Decisions and Recommendations of the Committee.

It was decided that we would continue our practice of determining the reaction to the Wassermann test in every patient with cancer of the mouth and, when the reaction was positive, give an intensive course of anti-syphilitic treatment for about two weeks prior to radiation therapy. During this period all infected teeth are to be extracted. In reasonably early tumours of the anterior two-thirds of the tongue we shall continue to implant radium needles in the tongue, while the glands will be treated three weeks later by a radical Crile's dissection whenever the patient's general health warrants it and the glandular invasion is not too great. If deposits are found in the dissected glands the area will receive post-operative deep X rays or radium mould. Tumours of the floor of the mouth will be treated by radium on a moulded apparatus or deep X rays or both. Tumours of the posterior portion of the tongue will be treated by combined interstitial treatment and deep X rays, or deep X rays alone in the more advanced lesions. We shall continue to use deep X rays for treating inoperable deposits in glands.

NOTICE.

AT the new Medical School of the University of Sydney a demonstration of urology has been arranged for medical students by the urologists on the staff of the Royal Prince Alfred Hospital and by the Department of Surgery. The demonstration represents a history of urology in the form of exhibits of a series of specimens, instruments and photographs. An invitation is extended to all members of the profession to inspect the demonstration, which will be showing for one month daily from 9 a.m. to 5 p.m.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx, xxi, xxii.

DEPARTMENT OF PUBLIC HEALTH, PERTH, WESTERN AUSTRALIA: Medical Officer.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.

HORNSBY AND DISTRICT HOSPITAL, HORNSBY, NEW SOUTH WALES: Relieving Resident Medical Officer, Honorary Officers.

INNISFAIL HOSPITAL BOARD, INNISFAIL, QUEENSLAND: Assistant Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Registrars.

ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, BRISBANE, QUEENSLAND: Honorary Officers.

THE PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Medical Officer.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistants, Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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